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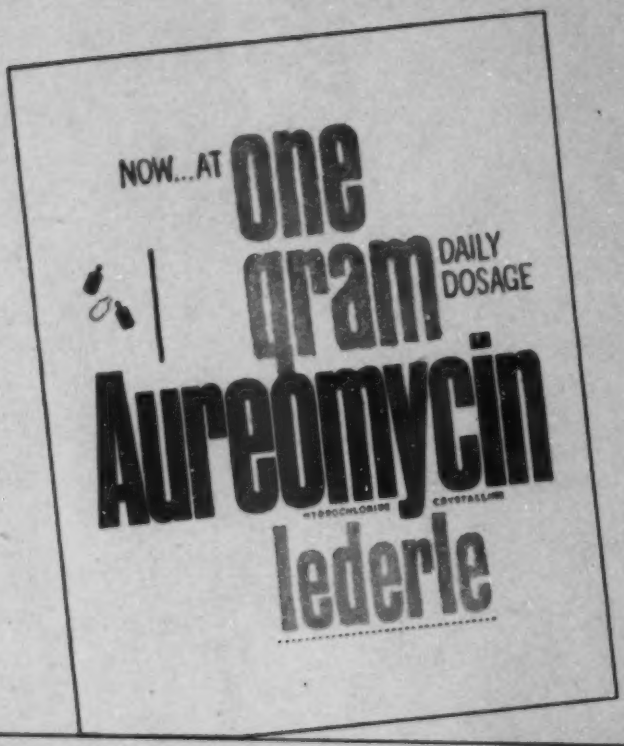


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## Contents.


**ORIGINAL ARTICLES :—** PAGE

**Digitalis in Cardiology.**—Dr. V. S. Prayag, M.D., F.C.P.S. (BOM.) Sassoon Hospitals and B. J. Medical College, Poona ... 665

**Vitamins and Detection of Their Early Deficiency**—S. Sankararaman, M.B., B.S., "Kumara Villa", Clive Street, Cuddalore Old Town .. 672

**The Epilepsies**—Miss. G. Bhattacharyya, B.Sc., M.B., B.S., Women and Children Hospital, Madras and S. Sen, B.Sc., M.B., M.R.C.P., (Lond.), F.R.F.P., & S. (Glas.), D.T.M. & H. (Lond.), Professor of Clinical Medicine, National Medical Institute, Calcutta ... 683

**Artificial Insemination — Its Medico-Legal and Social Aspects.**—D. J. Reddy, M.D., Prof. of Forensic Medicine, Andhra Medical College, Visakhapatnam .. 693

**Cœliac Disease**—A. V. S. Sarma, M.B., B.S., D.C.H. (Lond.), F.D.S. (Lond.), Honorary Physician, Govt. Royapettah Hospital, Madras. 697

**The Rhesus Antibodies**—Lt. Col. G. W. G. Bird, Lieut-Col[s] Army Medical Corps, Officer-in-charge, Blood Transfusion Department, Armed Forces Medical College, Poona. ... 701

**The Malaria Menace and How to Avert it**—P. N. Gupta, B.Sc., F.R.S.T.M. & H., M.R. San. I: (London), Station Hygiene Organisation, Pathankot Camp P.O., Dist. Gurdaspur (Punjab) 703

**CASES AND COMMENTS :—**

**Report on a Study of the Value of Antibiotics in Cholera (Terramycin, Chloromycetin and Aureomycin) and Formocibazol**—A. K. Roy Chaudhuri, M.D. (Cal.), Professor-in-charge, Department of Medicine, R. G. Kar Medical College Hospitals, Calcutta, Anil Chaudhury, M.Sc., M.B., D.T.M., Department of Pathology, R. G. Kar Medical College, Calcutta, and V. N. Chadha, M.B., (Cal.), Research Scholar, Department of Medicine, R. G. Kar Medical College Hospital, Calcutta. ... 717

PAGE

**Hepato-Splenomegaly of Undetermined Etiology—Lipoidoses ?**—A. V. S. Sarma, M.B., B.S., D.C.H. (Lond.), F.D.S. (Lond.). Honorary Physician, Government Royapettah Hospital, Madras. ... 725

**EDITORIALS :—**

**Child Welfare—Pædiatricians in Conference** .. 727

**Ministerial Pronouncements of Moment—(Conference of District Medical Officers)** ... 729

**The Training of Nurses—A New Experiment in Madras** ... 733

**GLEANINGS from MEDICAL PRESS :—**

**Medicine and Therapeutics**

Effective and economic treatment of acute bacterial pneumonia at home ... 682
Aneurin (thiamine hydrochloride) and intolerance to opiates ... 682
Aspirin and hæmatemesis ... 716
Return of vestibular function following streptomycin toxæmia ... 716
Terramycin in treatment of pneumonias of childhood ... 724
Aetiology of diabetes mellitus: The changing concept ... 736
Sulphonamides and antibiotics in amœbic dysentery ... 737
Resochin in amœbic hepatitis ... 737
Allergy to aspirin ... 738

**Surgery**

Re-establishing lymph drainage in lymphœdema ... 738
Surgical management of secondary hypersplenism ... 739
Control of bleeding in cranial operations ... 739
Rehabilitation after poliomyelitis ... 739

**REVIEWS OF BOOKS &c.,**

Bone Tumours .. 740
Pædiatric Allergy .. 740

**ACKNOWLEDGEMENT :** ... 740

**CORRESPONDENCE :** .. 741

**NEWS & NOTES :** ... 743

**OBITUARY :** .. 744



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## Index to Advertisers


| | PAGE |
|---|---|
| Agrawal & Co. Ltd. | .. 60 |
| Alarsin Pharmaceuticals (India) | .. 80 |
| Albert David Ltd. | 23, 54 |
| Alembic Chemical Works Co., Ltd. | .. 38 |
| Allen & Hanburys Ltd. | .. 31 |
| Alliance Trading Corporation | .. 76 |
| Amratlal & Co., B. | .. 76 |
| Angier Chemical Co. | .. 58 |
| Anglo-Thai Corporation Ltd. | .. 68 |
| Asepticus Co. | .. 78 |
| Associated Drug Co. Ltd. | .. 66 |
| Associated Electrical Industries (India) Ltd. | 13 |
| Atlantis (East) Ltd. | .. 9 |
| Attarwalla & Co. | .. 73 |
| B. A. Bros. | .. 81 |
| Behar Chemical Works | .. 81 |
| Bengal Chemical *Inside of Back Cover* | |
| Bengal Immunity Co. | .. 53 |
| Biddle Sawyer & Co. (India) Ltd. | .. 54 |
| Binod Mills Co. Ltd., The | .. 8 |
| Birla Laboratories | .. 67 |
| Boots Pure Drug Co. (India) Ltd. *Front Cover* & | 52 |
| Brahmachari Research Institute, The | .. 59 |
| Brand & Co. Ltd. | .. 29 |
| British Drug Houses (India) Ltd. | 44, 53 |
| Calcutta Chemical Co., Ltd. | 10, 16 |
| Calcutta Metallic Co. | 6 |
| Capco Ltd. | 42, 43 |
| Chatterjee, J. C. | .. 8 |
| Chowgule & Co. | .. 26 |
| Ciba Pharma Ltd. | .. 37 |
| Cilag-Hind Limited | .. 48 |
| Cipla Laboratories | .. 36 |
| Coates & Cooper Ltd. | .. 55 |
| Continental Drug Stores | .. 81 |
| Crookes Laboratories Ltd. | .. 86 |
| Current Technical Literature Co. Ltd. | 3, 6 |
| DeChane, J & J | .. 67 |
| Diamelin Research Laboratory | .. 69 |
| Dragon Chemical Works (Research) Ltd. | .. 77 |
| East Asiatic Co. (India) Ltd. insertion P. | 51, 11 |
| Eastern Surgical Company | .. 8 |
| East India Pharma. Works Ltd. | .. 49 |
| Eli Lilly & Co. | .. 34 |
| Fedco Ltd. Insertion P. | 42 |
| G. D. A. Chemicals Ltd. | .. 20 |
| Glaxo Laboratories Ltd. | 15, 21 |
| Gluconate Ltd. | .. 71 |
| Gordhandas Desai & Co. | .. 63 |
| Grahams Trading Co. (India) Ltd. | .. 76 |
| Hering & Kent | .. 68 |
| Himalaya Drug Co. | .. 78 |
| Hind Chemicals Ltd. | 51, 72 |
| Horlicks | .. 22 |
| Howards & Sons Ltd. | .. 47 |
| Imperial Chemical Industries (India) Ltd. | 5 |
| Imperial Surgical Co. | .. 75 |
| Indian Chemical & Therapeutical Works Ltd. | 50 |
| Indian Schering Limited | .. 24 |
| Indoco Remedies Ltd. | .. 81 |
| Indo-French Pharmaceutical Co. | 57, 60 |

| | PAGE |
|---|---|
| Indo-Pharma Pharmaceutical Works | .. 66 |
| Jai Singh & Co. Dr. | .. 6 |
| Jammi Venkataramanayya & Sons | .. 58 |
| John Wyeth & Brother Ltd. | 25, 35 |
| Juggat Singh's Son & Bros. | 16, 50 |
| Kothari Book Depot, The | .. 7 |
| Lederle Labs. (India) Ltd. *Inside of Front Cover* | |
| Mandoss & Co., Ltd. | .. 75 |
| Martin H. Smith Company | .. 55 |
| May & Baker (India) Ltd. *Front Cover* & | 39 |
| Merck (North America) Inc. | .. 74 |
| Model Pharmacy | .. 75 |
| Morison, Son & Jones (India) Ltd., J.L. | .. 76 |
| Mukerji & Banerjee Surgical Ltd., | 70, 77 |
| Narindar S. Uberoi | .. 82 |
| Nath & Co. | .. 85 |
| Neo-Pharma Ltd. | 10, 23 |
| Nestles Products (India) Ltd. | .. 27 |
| New Scientific Mart | .. 6 |
| New Surgical Trading Co., The | .. 12 |
| Opil Ltd. | .. 19 |
| Organon Laboratories Ltd. | .. 17 |
| Oriental Research & Chemical Lab. Ltd. | 61, 64 |
| Ovaltine (Grahams Trading Co.) | .. 32 |
| Parke, Davis & Co. *Outside of Back Cover* | |
| Pasteur Laboratories Ltd. | .. 65 |
| Pfizer | .. 18 |
| Pharmed Ltd. | .. 41 |
| Philips Electrical Co. (India) Ltd. | .. 40 |
| Phœnix Drug House | .. 64 |
| Pixie Products | .. 78 |
| Popular Book Depot, The | .. 7 |
| Pravin Laboratory | .. 65 |
| Primco Limited | .. 63 |
| Sandoz Ltd. Insertion P. | 33, 46 |
| Sarabhai Chemicals | 45, 70 |
| Sarker & Co., N. R. | .. 8 |
| Scientific Publishing Co. | .. 3 |
| Scientific Publication Concern | .. 7 |
| Shah & Co., D. | .. 6 |
| Shanti Trading Co. | .. 79 |
| Smith Kline & French International Co. | .. 41 |
| Smith Stanistreet & Co., Ltd. | 14, 51 |
| Sri Krishnan Bros. | .. 2 |
| Standard Pharmaceutical Ltd. | .. 61 |
| Suren & Co., Ltd., W.T. | .. 28 |
| Swann & Co., Ltd., W. R. | .. 9 |
| Tropical Chemical Works | .. 77 |
| Union Drug Co., Ltd. | .. 69 |
| Unique Trading Corporation | .. 71 |
| Universal Drug House Ltd | .. 77 |
| Unjha Pharmacy Limited | .. 84 |
| Universal Pharmaceutical Works Ltd. | .. 78 |
| Universal Traders A.M. | .. 8 |
| Volkart Bros. | .. 33 |
| Wander Pharmaceutical Department | 1, 30 |
| Ward, Blenkinsop & Co. Ltd. | .. 3 |
| Waterbury's Compound | .. 56 |
| William R. Warner & Co. | 22, 57, 72 |
| Wincarnis | .. 56 |
| Worli Chemical Works Ltd. | .. 59 |
| Zandu Pharmaceutical Works Ltd. | .. 62 |
| Zill & Co. | .. 83 |
| Zone Chemical Co | .. 62 |

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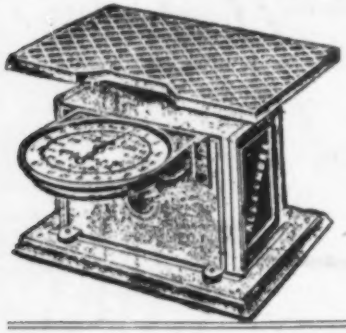

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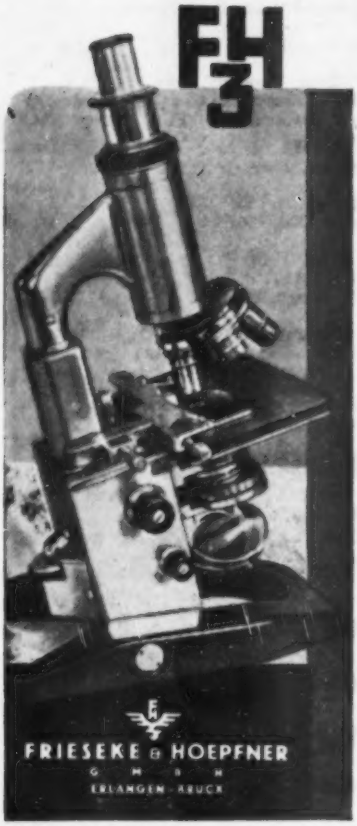


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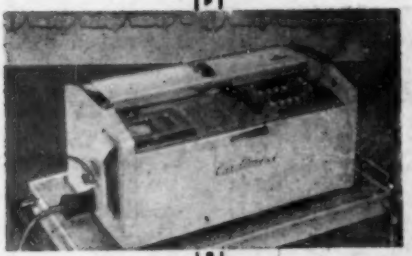

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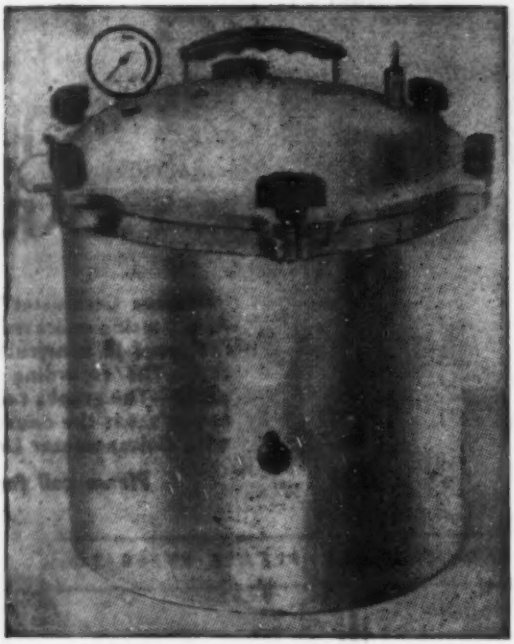

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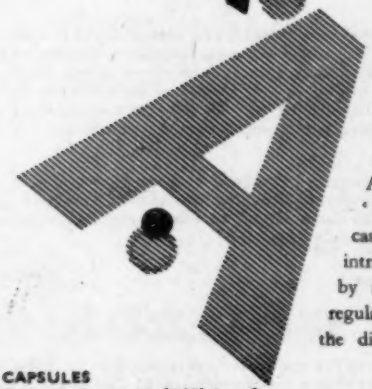


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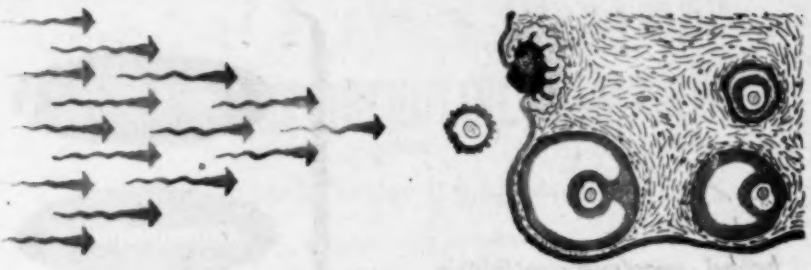

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1. Tobie, J. E.; Most, H.; Reardon, L. V., and Bosicevich, J.: Am. J. Trop. Med. 31:414 (July) 1951. 2. King, E.Q., et al.: J.A.M.A. 143:1-4 (May 6) 1950.

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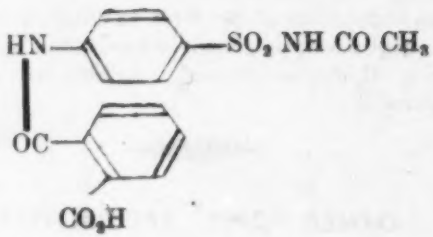


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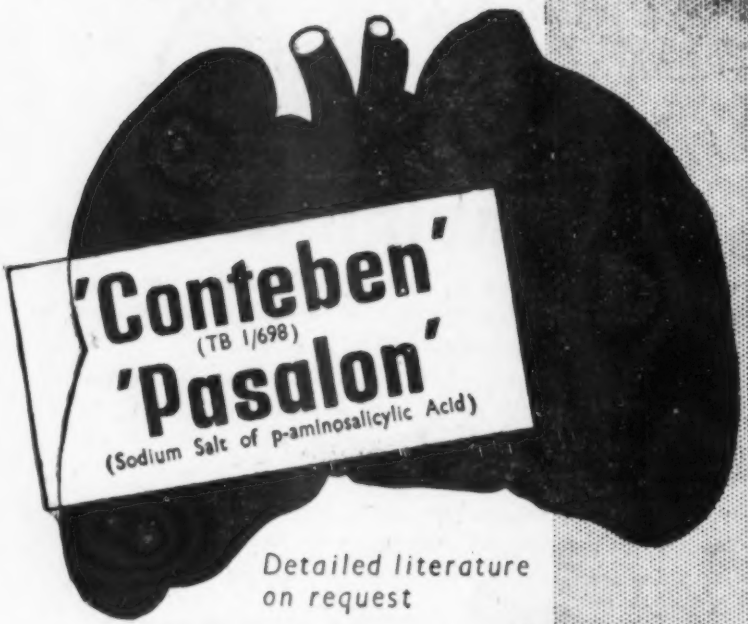


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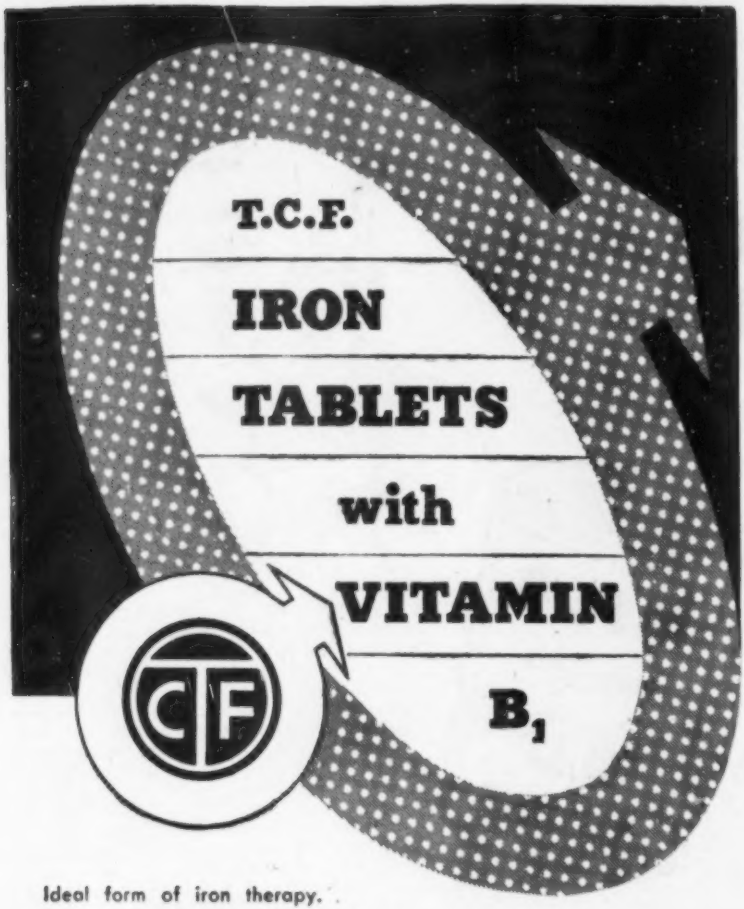


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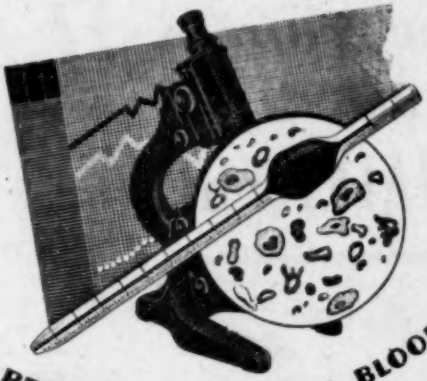

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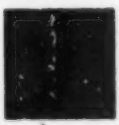

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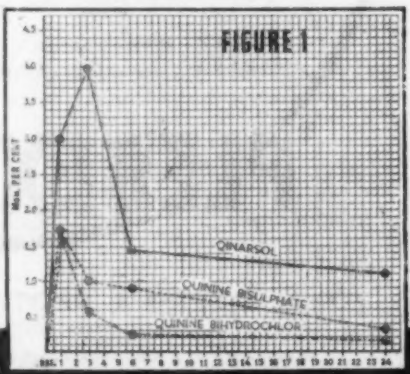
FIGURE 1

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## Original Articles

# DIGITALIS IN CARDIOLOGY*


DR. V. S. PRAYAG, M.D., F.C.P.S. (BOM.),

*Sassoon Hospitals and B. J. Medical College, Poona.*


Introduction.—Digitalis is one of the few drugs which have proved to be very effective and even indispensable in modern therapy. Used over a long period, the treatment by this drug in diseases of the heart has undergone several changes recently and as William Dock[1] says "the growth of our knowledge of digitalis is so steady and important that every physician who wishes to use these substances effectively and intelligently needs to review the subject every few years in a thorough and painstaking fashion." In the art of medical treatment, the drug holds a place similar to liver extract in pernicious anæmia and the recent introduction of lanatoside C (perhaps similar to vitamin $B_{12}$ to continue the analogy) has been a further step forward in therapeutics.

*Historical*:—Named so, because of the resemblance of the flower of the plant to a finger, digitalis purpurea was first recognised by Fauscius in 1542; however, not until William Withering[2] first demonstrated them, were the diuretic properties known. He became interested on studying a number of secret remedies used by an old Shropshire woman, who had successfully used it for treating a case of generalised anasarca. He wrote down his observations and published them after 12 years; in doing so he had not failed to note the effect of foxglove on the pulse of the patient.

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After many years during which the drug had been practically forgotten, James Mackenzie[3] and Thomas Lewis rediscovered its remarkable effect on a case of auricular fibrillation. The slowing of the heart-rate in this condition made them believe that is was the only predominant action of the drug and any relief the patient obtained was due to its action on arrhythmias—and this wat the only indication for digitalis. Even today, some physicians believe that the drug is to be used only for disturbances of cardiac rhythm.

Pharmacology.—There are two species of the digitalis plant:—the lanata and the purpurea—the latter being the source of the official preparations. The drug is obtained from the seeds and leaves of digitalis purpurea and contains certain active ingredients called glycosides. The lanata contains digitoxin, gitoxin and digoxin and the purpurea gives the first two and a third alkaliod called gitalin.

*Action on the heart*:—This is the most significant action of the drug for which it is chiefly used in medicine. It is truly called a cardiotonic drug—since it greatly increases the force of systolic contraction of the heart-muscle. It also increases the tone of the myocardium and the irritability of the muscle. By increasing the force of contraction, the heart is better filled, especially the auricles during diastoles. The efficiency with which the heart pumps blood thus increases, with a resultant increase in the cardiac output. Mackenzie, Lewis and Fishberg[4] have rightly emphasised the action of this drug on the rate of heart-beat and on increasing the rest-period. The heart-rate is affected both in health and in disease and the action is noted equally in irregular as well as in regular rhythm. Cushney and Mackenzie[5] were of the view that the slowing of the heart beat was caused by a partial auriculo-ventricular block. Later on, it was proved that the drug also acted on the myocardium besides the A. V. node. The role played in vagal stimulation should not be forgotten. Digitalis, in the initial stages stimulates the vagal centre and the vagal nerve endings and thus slows down the heart. But later on, the action is directly on the myocardium, independent of the vagal action and is not abolished by large doses of atropine.

It has been noted that the onset of action of the various glycosides differs with different active glycosides and Rothlin [6] believes this to be due to the property of various glycosides getting adsorbed to the proteins of blood.

*Cardiac output*:—Cushney, Cohn and Steele[7] and McMichael and Sharpey-Schafer [8] have all found a definite increase in the cardiac output–whenever the latter was low due to congestive failure. With an increase in the force of systolic contraction, the ventricles are emptied more completely and efficiently and thus the output is increased inspite of a slow rate.

*Venous pressure*:—The venous pressure is considerably raised in congestive heart failure and digitalis, by relieving congestion

in various parts of the body and increasing the cardiac output, decreases the venous pressure.

*Diuretic action* :—Since Withering's time, the digitalis group of drugs has been known to act as diuretics in dropsy due to heart disease. This action which is, however, indirect is due mainly to an improvement in circulation enabling more blood to go to the kidneys. The kidneys are thus able to filter a larger quantity of blood and hence diuresis takes place. Therefore, in œdemas of non-cardiac origin and in other oligurias, digitalis will be of no avail and should not be used. The urinary output increases considerably in some cases especially within the first few days and thus relieves the œdema. However, some cases are refractory requiring other and more powerful diuretic drugs like Mersalyl, Theobromine or Theophylline along with the cardiotonic drug.

*Toxic effects* :—Digitalis, in any form is not a safe drug. In fact, as stated by Goodman and Gillman, [9] the toxicity may be regarded as an extension of the therapeutic action of the drug. Different preparations differ in their degrees of toxicity, but an "entirely non-toxic digitalis does not exist".

As part of the drug remains in the body for a long time, accumulation takes place giving rise to serious effects. These should, therefore, be carefully looked for, to avoid catastrophes. Withering who had observed these reactions states, "It is well to remember to continue the drug, until either it acts on the kidneys, the stomach, the pulse or the bowels; let it be stopped upon the first appearance of any one of these effects". Movitt [17] states that this advice is frequently overlooked, as statistics show that the *incidence of toxic cases is increasing every year*. The toxic effects consist of a complete loss of appetite, nausea and sometimes vomiting which may be severe. This may also be due to congestion of the stomach and is not therefore, an indication for stopping the drug. When it does occur, and if the condition demands treatment with digitalis, a different preparation or brand may be tried. Headache may occasionally be so intense as to make it impossible for the drug to be continued. Cardiac manifestations are fairly common. Extra beats or premature contractions are among the commonest. Digitalis sometimes causes coupling or bigaminal type of pulse when the drug should be withdrawn immediately. The heart rate may become dangerously slow—below 45 or 50 when a further continuation of therapy should not be hazarded. The pulse-rate should be watched from day-to-day and abnormalities noted very carefully.

Sampson and his co-workers [9] have shown that the ectopic beats caused by digitalis can be abolished in every instance by the oral administration of potassium salts. They conclude that the disturbance of the potassium balance is related to toxic doses of digitalis.

Digitalis itself in toxic doses, might lead to auricular fibrillation, though it is used for treating that condition. The kidneys also manifest toxicity by a lowered output of urine when large doses are used over a long period. Very rarely, anuria may occur. Hueper [10] has summarized some of the toxic effects of digitalis in a recent report. He points out that the correct evaluation of symptoms due to digitalis-poisoning may be difficult as they may simulate some of the symptoms of the heart disease itself. The absorption of digitalis and its preparations takes place through the intestines, part of this drug getting destroyed in the liver. The rate of absorption varies with different preparations, digitoxin being almost completely absorbed. The excretion is not fully understood, though a part is excreted through the kidney and a part detoxicated in the liver. Cumulative effects varies with different preparations.

*Standardization* :—Different specimens of digitalis purpurea contain varying porportions of the active principles, because of the different areas from where it is obtained and also because specimens from the same area may vary in potency according to the age of the plant ; so the necessity for standardization is imperative. However, as the chemistry of the active principles is not yet fully understood, to allow standardization by chemical analysis, we resort to biological methods of assay. Various animals were tried but the frog and more recently the cat-methods are widely used at present. The systolic arrest of the cat's heart in one hour is taken as the end point. One U.S.P. unit of digitalis or the International Digitalis Unit is the equivalent of 0·1 gm. of the International Standard Digitalis powder. Carter[11] stresses that "in view of the wide variations in potency of different products even allegedly assayed in accordance with one and the same standard, the clinician must depend on the presumably equal potency of the same manufacturer's product. He should urge each patient to use a certain product exclusively." In this connection, it may be stated that lanatoside C can be assayed fairly accurately by chemical methods and hence should be the drug of choice whenever exact quantities have to be administered.

Lanatoside C.—Digitalis purpurea and lanata have each three glycosides but only the latter contains lanatoside C ; and a combination of the three lanatosides, *viz.*, A, B & C is termed Digilanid. Stoll [12] isolated from the leaves of digitalis lanata these lanatosides and proved that the pure glycosides, digitoxin and gitoxin are actually degradation products of the compounds as they exist in the natural state. Lanatoside C is a pure chemical substance readily soluble in alcohol, but only slightly in water. Moe and Visscher [13] state "of the three lanatosides C is the least toxic and has the maximum therapeutic action/toxicity ratio." La Due and Fahr [14] in their study of lanatoside C on cardiac cases found that it decreased the venous pressure and increased

the pulse-pressure and also the mechanical efficiency of the heart. They concluded that it was definitely less toxic than preparations of digitalis purpurea. Kwitt, Gold and Catell[15] found that it produced digitalis-like changes in the electro-cardiogram. Lanatoside C has a strophanthin-like action in that it affords prompt and certain relief in congestive heart failure and auricular-fibrillation with the added advantage that it can be given safely by mouth as well as by the intravenous route. Fahr and La Due [14] found the drug very useful in various arrhythmias like auricular fibrillation, flutter and paroxysmal tachycardia. In the last named condition lanatoside C appears to be far superior to any other drug. In one of my *own* cases of paroxysmal tachycardia which was persistent for 3 days and where the electro-cardiogram confirmed the diagnosis, the drug (Cedilanid-Sandoz) was found to be effective and worked within 6 hours when all other methods of treatment had failed. A similar case is mentioned by Movitt [17] who treated auricular flutter successfully by heroic doses of lanatoside C (4 tablets 4 times a day) when large doses of digitalis and quinidine had failed. White[16] refers to lanatoside C as a reliable glycoside and states that "a very convenient and highly satisfactory way to digitalize rapidly is to use ampoules of Cedilanid." Lanatoside C can be standardized by the gravimetric method—a decided advantage over glycosides which have to be standardized by the unreliable and difficult method of bio-assay. Lanatoside C is a stable, easily absorbed and effective preparation. It has all the indications of digitalis; it is more effective than other lanatosides and owing to its greater speed of elimination is less toxic. It can be safely given by the intravenous route when rapidity of action and parenteral administration are considered necessary.

Therapeutics.—Digitalis is an excellent drug for use in cases of congestive heart failure—especially the low out-put failure and the left sided heart failure or cardiac asthma. The drug seems to act almost specifically and produce remarkable relief in some cases. Whatever the cause, digitalis should be used as early as feasible in all cases of congestive failure. Partial or complete heart-block, coronary sclerosis—anginal attacks, etc., are not contra-indications, where there is positive evidence of decompensation. Among various disturbances of rhythm, auricular flutter and fibrillation seem to respond well to digitalis. Lanatoside C which can be safely administered intravenously is effective in bringing down the abnormal rate to a normal range in from a few minutes to a few hours. The drug is very useful in these arrhythmias complicated by congestive failure. The treatment by digitalis should not be delayed till the classical signs of heart failure have appeared. It is wise to start the drug as early as possible, whenever the first signs and symptoms of failure are observed. White and Christian[16] believe in the therapy being started even earlier—*viz.*, in enlargement

of the heart with hypertension or valvular disease, even without evidence of failure. In cardiac asthma due to aortic incompetence or other valvular disease and to hypertension, digitalis is again the drug of choice.

Dosage.—It is essential to remember that the dosage of any preparation of digitalis depends entirely on the individual needs, and hence no rigid rules can be fixed. A few points are, however, worth noting :

White [16] makes the following recommendations :

> In case of urgency 0·3 gm. of powdered leaves 8 hourly for one day.
>
> In case of less urgency 0·2 gm. of powdered leaves 8 hourly for two days.
>
> In case of no urgency (patient ambulant) 0·1 gm. of powdered leaves thrice a day for 5 to 7 days.

For convenience it is necessary to know the corresponding quantities of the different preparations. These are :—0·1 gm. of powdered leaf is equivalent to 15 min. of Tincture or 0·25 mg. of Digoxin. For rapid digitalization the intravenous route is usually preferred ; for this purpose, strophanthin, digitoxin and lanatoside C may be used. For routine therapy, I prefer giving 2 to 4 tablets of digoxin (0·50 to 1·0 mgm.) as the first dose and one tablet every 6 to 8 hours according to the needs of the case, on the first day. Subsequently one tablet thrice a day, reduced gradually to one per day as a maintenance dose. As Luten says, "digitalis dosage is accurately to be measured not in terms of the weight or volume of the preparation employed, nor indeed in biological units, but in terms of the effect produced".

*Contra-indications* :—Digitalis is very useful in paroxysmal tachycardia of auricular origin ; but similar tachycardia of ventricular origin is an absolute contra-indication, as it converts the condition into ventricular fibrillation which is very dangerous and may prove rapidly fatal. The irrational use of digitalis should be avoided. For instance, it will be of no use in circulatory failure due to shock or peripheral vaso-dilatation. Similarly any rapid pulse will not respond to digitalis. It is futile to give digitalis to a case of thyrotoxicosis with a rapid pulse and a well-compensated heart. In high fevers, digitalis should be avoided as far as possible, as it is ineffective and may further irritate the myocardium. Coronary thrombosis without any evidence of cardiac failure is a contra-indication for digitalis as the drug may aggravate the condition.

Cardiac failure due to conditions like beri-beri etc., are likely to respond better to therapy aimed at removing the cause ; hence digitalis should be avoided in these cases also. When digitalis is being given to a patient, calcium should be withheld as far as possible —as cases of death have been reported, following intravenous calcium in a previously digitalized patient (Bower and Mengle [18]).

Choice of preparations.—A wide range of digitalis preparations is available, each having its own distinctive place and use in cardiac therapy. Batterman and DeGraff[19] in a comparative study of digoxin, digitoxin and lanatoside C pronounced in favour of *digoxin*. (Digoxin is used more extensively in England than in America.) "Because of its slow dissipation and possibility of prolonged and severe toxicity, digitoxin is not the drug of choice." Master [20] concludes that "digitoxin poisoning has now become so frequent in occurrence that it presents a real hazard."

On the contrary, Levine[21] who made an extensive comparative study of digitalis leaf and digitoxin opined that it cannot be stated as a positive fact that digitoxin has a greater tendency than digitalis to produce toxic reactions."

Lanatoside C has been already considered *supra*. Digitalis leaf has one draw-back *viz.*, the powder is irregularly absorbed *via* the intestine, hence the dose cannot be adjusted accurately.

As the tincture loses potency in keeping, only fresh tinctures should be used.

Summary.—A review of the literature on the digitalis group of drugs and their place in cardiology is given as the fairly rapid advances being made, merit the focussing of our attention on the subject. An attempt has been made to present the various aspects relating to the use of this important drug in modern therapy.

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# VITAMINS AND DETECTION OF THEIR EARLY DEFICIENCY *

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VITAMINS are essential accessory food factors and can be classified with regard to human needs into three categories:

(1) *Vitamins of undoubted clinical significance*:—These include Vitamin $A_1$, Vitamin $B_1$, four of the Vitamin B-complex, *viz.*, nicotinic acid, riboflavin, folic acid and $B_{12}$, Vitamin C, Vitamin D and Vitamin K.

(2) *Vitamins of doubtful clinical significance*:—These include two of the Vitamin $B_2$ Complex i.e. biotin (Vit. H) and choline and also Vitamin E, Vit. F and Vit. P.

(3) *Vitamins whose significance has been demonstrated only in animal experiments*:—These include four of the vitamin $B_2$ group *i.e.*, pyridoxin, pantothenic acid, inositol and p. amino-benzoic acid.

Vitamin 'A' includes vitamin $A_1$ or axerophthol and the principal provitamin, *beta*-carotene. There are also several other forms of vitamin A *viz.*, vitamin $A_2$, anhydro vitamin A, sub-vitamin A, *alpha* and *gamma*-xanthene, aphanene and aphanicene. But these have no significance with regard to human requirements. Vitamin A is a pale yellow crystal with a molecular formula $C_{20}H_{30}$ and a melting point of 634° C. It was isolated in 1937 and synthesised in 1945.

*The signs of deficiency* are: diminished powers of dark adaptation which is the earliest and most delicate sign and which progresses on to night blindness ultimately, xerosis and keratinisation of the various membrane tissue, a keratinised follicular papular eruption on the skin, xerosis of the conjunctiva and cornea leading to biotic spots, xerophthalmia and in advanced cases to keratomalacia. In experimental animals, renal calculi, degenerative changes in the C.N.S., periodontal hyperplasia overgrowth of bone, failure of reproduction and impaired growth rate are present.

*The best sources of Vitamin A*: include halibut liver oil, cod liver oil, mammalian liver and milk. It occurs abundantly as carotene in green leafy vegetables and carrots. It occurs in eggs and butter both as vitamin A and as carotene. The League of Nations standard of requirement is 3000 I.U. (1 I.U. is equal to 0·6 microgram of *beta*-carotene) only one third to half of the carotene is absorbed by the animal body, as compared with an equal amount of vitamin A. The daily requirements of an active man or woman and a pregnant or nursing mother varies from 5000 to 8000 I.U.

* Specially contributed to THE ANTISEPTIC.

Vitamin B and its complex.—Vitamin $B_1$ is heat-labile and vitamin $B_2$ (complex of vitamin B) is heat-stable. Vitamin $B_1$, also called 'aneurin hydrochloride' or 'thiamin hydrochloride' is a white crystal with a nutty smell. a melting point of 250°C, and a molecular formula of $C_{12}$ $H_{17}O$ $N_4$ $C_1$ Hcl. It was isolated in 1931 and synthesised in 1936. Deficiency causes beri beri. It is common among people subsisting on polished rice and is characterised by polyneuritis of the peripheral nerves, manifesting itself as patches of parasthesia in the lower limbs, diminished knee-jerks, wrist drop and high stepping gait and also by cardiac irregularities such as shortness of breath, dyspnœa, tachycardia and a dilated right side of the heart, as also by a muscular weakness, anorexia and sometimes emaciation as in dry beriberi, or œdema as in wet beriberi. It is well to bear in mind that the disease may manifest itself in many different forms depending on whether the polyneuritic, cardiac or œdematous symptoms predominate. Conditioned deficiency of vitamin B occurs in polyneuritis due to gastro-intestinal obstruction, in alcoholics and in pregnancy owing to faulty absorption of this vitamin. In infants, sub-optimal intake leads to inadequate growth and gastro-intestinal hypotonia. The League of Nations standard of requirement in 300 I.U. per day (1 I. U. is equal to 3 mg.). *The best sources of this vitamin* are: yeast, whole cereals, pork, liver, nuts, eggs, legumes and wheat germ.

Vitamin $B_2$.—When vitamin $B_2$ was first recognised it was described "as the heat-stable water-soluble fraction present in extracts of yeast, preventing pellagra in man, black-tongue in dogs and pellagra-like dermatitis in rats, and needed for the promotion of health and growth in rats." This was subsequently found to include the following complexes: (1) nicotinamide; (2) riboflavin; (3) pyridoxin; (4) pantothenic acid; (5) p. amino-benzoic acid; (6) inositol; (7) biotin; (8) choline; (9) folic acid; and (10) vitamin $B_{12}$.

Riboflavin ($B_2$) is an orange coloured crystal with a melting point of 282°C; is fluorescent and sensitive to light. It occurs in living tissues combined as a co-enzyme of flavoproteins *i.e.* iso-alloxazine adenine di-nucleotide. Deficiency of the vitamin causes cheilosis (lesion of the lips) and angular stomatitis. There are cracks, scaliness and maceration at the corners of the mouth and fissures appear later. Opacity and vascularisation of the cornea are also described. There is an eczematous condition and itching of the scrotal and penis integument. There is no international standard unit for the same. The daily requirement is about 1 to 2 mg. *The best sources of this vitamin are*: yeast, liver, kidney, milk, white of eggs, green vegetables and peas.

Pantothenic acid $B_3$.—Formerly called chicken-pellagra-factor or filter-factor, occurs as a pale yellow oil. Its calcium salt is a white crystal. It has a molecular formula of $C_9H_{17}O_5N$. It prevents

dermatitis in birds and assists growth in rats. Its absence causes dermatitis, nose-bleeding, depilation about the nose, sticky exudate in the eye-lids and achromotrichia. It is also a growth factor for micro-organisms. Its significance as a factor in human nutrition is still open to doubt.

Nicotinamide $B_5$.—Otherwise called nicotinic amide or niacin amide or pellagra-preventing-vitamin, it is a white crystal with a melting point of 235°C, and a molecular formula of $C_6H_5O_2N$. Nicotinamide occurs in living tissues as (1) co-dehydrogenase I or 'Euler's Cozymase' di-phosphopyridine nucleotide; and (2) co-dehydrogenase II, or 'Warburg's enzyme' tri-phosphopyridine nucleotide. *The sign of deficiency* of this vitamin is pellagra with its characteristic glossitis, stomatitis, diarrhœa and pigmentation of the skin which is bilaterally symmetrical and which occurs on the parts of the body exposed to the sun or to friction. Secondary pellagra or conditioned deficiency of nicotinamide is caused by faulty absorption or assimilation of the vitamin as in chronic alcoholism and gastro-intestinal obstruction. There is no international unit prescribed but the daily requirement is assessed at 10 to 20 mg. *The best sources of this vitamin are* : yeast, liver and liver extracts, fish, especially canned salmon, meat, pulses, whole wheat-meal or bran and green vegetables.

Pyridoxin $B_6$ known also as 'adermin' is a white crystal with a melting point of 160°C and a molecular formula of $C_8H_4O_3N$. This vitamin prevents a disease in rats characterised by florid symmetrical dermatitis of the extremities. It is stated to be concerned in the synthesis of fats from protein and perhaps in tryptophan metabolism. Its need for human nutrition is still undetermined but has been found useful in the treatment of agranulocytosis.

P-amino-benzoic acid occurs as white crystals with a melting point of 186°C, and is a growth factor for various micro-organisms. It is only a secondary nutrient for man. It has been proved that the sulpha group of drugs exert their bacteriostatic action by blocking the supply of PAB acid to the organisms.

Inositol occurs as white sweetish crystals with a melting point of 225°C, and a molecular formula of $C_6H_{12}O_6$. It is useful for the growth of yeast and other micro-organisms. It causes alopecia in rats. It is useful only as a secondary nutrient to man by stimulating the intestinal flora to synthesise other nutrients essential to human beings.

Biotin is a white crystal with a melting point of 230°C. and is identical with vitamin H (huatskin factor) and anti-egg white injury factor for rats and other animals. Its significance in human diet is not yet sufficiently known.

Choline is chemically a trans-methylating agent and is replaceable by the amino-acid, methionine or by betaine. The deficiency

of this vitamin results in fatty liver and kidney hæmorrhages. Its clinical significance is to control the negative nitrogen balance or in other words, it is as effective as a high protein diet.

Folic acid is also known as grass-juice factor for guinea pigs vitamin Bg (anti-anæmic factor for chicks) vitamin M. for monkeys, and the growth factor for the micro-organism *lacto-bacillus casii* and for streptococcus lactis R. It is an orange-yellow crystal with the molecular formula $C_{19}H_{19}O_6N_7$. It is built from glutonic acid, p-amino-benzoic acid and pterin residues. It produces a hæmatopoietic response in Addisonian pernicious anæmia and in some other macrocytic anæmias in man. It is useful in the treatment of sprue. It does not however, prevent degeneration of the cord in pernicious anæmia. Its anti-carcinogenic action is in the experimental stage. The best sources of this vitamin are liver, yeast, green leaves and spinach.

Vitamin $B_{12}$ is a red cobalt-containing crystalline substance with a molecular weight of about 1600. It is the treatment of choice in pernicious anæmia. A single injection of 3 to 6 micro-grams produces a maximal reticulocyte response and a total dose of 56 micrograms is all that is necessary to maintain hæmatological response. But in patients with neurological lesions 1 microgram of vitamin $B_{12}$ a day for fifty-three days and then 25 micrograms a week is necessary for the neurological signs to disappear. Bethell reported that the response to vitamin $B_{12}$ is inhibited by folic acid antagonists. Folic acid is needed by almost all living cells. Hence if folic acid is blocked by a folic-acid-antagonist in a patient with megaloblastic anæmia, the administration of vitamin $B_{12}$ becomes ineffective in restoring the bone-marrow to normal.

Vitamin C.—Known as l-ascorbic acid and as cevitamic acid it is the anti-scorbutic vitamin; it is a white crystal with a melting point of 190°C, and the molecular formula of $C_6H_8O_6$. *Deficiency of this vitamin produces*: scurvy characterised by spongy gums, petechial hæmorrhages in the skin of the trunk and limbs. The earliest sign is a follicular hyper-keratosis. There is lassitude, sallowness, mental depression and pains in the lower limbs, and also a hardening of the calf-muscle due to hæmorrhage. In infants, there is such tenderness of the lower limbs that the child dreads touch; subperiosteal hæmorrhage with swelling near the lower end of the femur and the long bones show a cessation of osteogenesis. The vitamin C content can be estimated in the urine by chemical tests (titration with dichlorphenol-indophenol). The League of Nations standard requirement for humans, is 30 mg. per day. *The best sources of this vitamin* are: fresh fruits, black currants, strawberries, oranges, lemon, grape fruit, gooseberries, *nellikkai* and tomatoes. It is also present in cauliflower, cabbage and fresh potatoes. The loss of vitamin C in cooking can be prevented by using small amounts of water for cooking, by not prolonging the time of heating

and not keeping them hot for a long time and not re-cooking before serving. This vitamin is useful in the treatment of wounds, especially fractures.

Vitamin D is the anti-rachitic vitamin. Vitamin $D_2$—calciferol or irradiated ergosterol is prepared artificially by ultra-violet irradiation of ergosterol. It occurs as a white crystal with a melting point of 115°C. Vitamin $D_3$ or irradiated dehydrocholesterol (chole-stadien-3-ol) is present in fish liver (halibut and cod) oils, in butter, eggs, cream and liver. Other D vitamins are irradiated cholesterol, cholesterilene, sulphonic acid, irradiated 22-dihydroergosterol and irradiated dehydrositosterol. Deficiency of this vitamin is usually stated to be responsible for the deficient calcification of bones especially at the epiphyses. The zone of provisional calcification is increased in breadth and inadequately mineralised. The consequent irregular growth of soft osteoid tissue causes the swelling at the epiphyseal junctions. The hardening of the inadequately calcified bones and the overgrowth of the osteoid tissue gives rise to bow and bandy legs, rachitic pelvis, enlargement of the ends of the bones, bending of the ribs resulting in pigeon breast and rickety rosary.

Absence of vitamin D was considered the main recognised cause of rickets. The factors at work include the ratio between calcium and phosphorus in the body. If the ratio is not correct, Vitamin D will correct it. When this ratio is correct and no vitamin D is present, ricket occurs. But when rats kept on vitamin D or calcium and phosphorus are exposed to ultra-violet rays, rickets do not develop. If the diet is absolutely deficient in vitamin D, or calcium and phosphorus, ultra-violet rays will not prevent the formation of rickets. Ultra-violet rays only correct and rectify a relative deficiency in the above constituents of food. So, the fundamental lesion in vitamin D deficiency is excessive loss of phosphate and/or calcium in the fæces with a concurrent drop in their levels in the blood and consequent imperfect calcification of bony tissues. This deficiency in the blood of inorganic phosphate and sometimes also of calcium causes tetany. Osteomalacia and abnormal dentition result from vitamin D deficiency. The international standard requirement for children is 500 to 1500 I. U. per day (protective) 3000 I. U. per day (curative). One I.U.=0·02 mg. of calciferol. The best sources of this vitamin are fish liver oil (both shark and cod) butter, eggs, cream and liver. Vitamin D is used to maintain the level of the blood calcium after parathyroidectomy and in the treatment of lupus vulgaris, by massive local injections.

Vitamin E includes *alpha*-tocopheral, *beta*-tocopherol, *gamma*-tocopherol and several related synthetic derivatives. It occurs as a light yellow oil with the molecular formula of $C_{29}H_{50}O_2$. Its absence produces in experimental animals:—(1) reproductive failure

due, in the female to resorption of the fœtuses during gestation and in the male, to degeneration of the germ cell; (2) muscular dystrophy; (3) degeneration of the muscle fibres leading to discolouration of the uterus and other tissues; (4) degeneration of the epithelium of the convoluted tubules of the kidney; (5) an exudative diathesis; and (6) dental depigmentation. It is claimed to be a fertility-factor in human beings and also a preventive of habitual abortion in women; and it is also indicated in cases of muscular dystrophies. Proof of the effectiveness of vitamin E is lacking as judged by controlled experiments and its use is merely based on a matter of hope. The best sources of this vitamin are wheat germ oil, rice germ oil, green vegetables and cotton seed oil.

Vitamin F is synonymous with the nutritionally essential unsaturated fatty acids *i.e.*, linoleic acid, linoleic and arachidonic acid. Deficiency of this vitamin causes a scaliness of the tail leading to caudal necrosis, irregularities in reproduction and kidney lesions in rats. In human beings it is claimed to be useful in eczematous conditions.

Vitamin P is a group of substances which include citrin (hesperidine glucoside). It has not been isolated in its pure state. It is claimed to control the fragility of the capillary blood vessels and is required as an adjuvant to vitamin C in the cure of some of the symptoms associated with scurvy, but experimental confirmation is lacking. The best sources of this vitamin are grapes and lemons.

Vitamin K.—Includes vitamin $K_1$ and vitamin $K_2$ and is an yellow oil having the molecular formula of $C_3H_{46}O_2$ ($K_1$ is 2-methyl 3-phytyl 1-naphthoquinone) and ($K_2$ is 2-methyl 3-diparanesyl 1-naphthoquinone). Other homologues are phthiocol and the parent substance 2-methyl-1 4-naphthoquinone. The latter is of easy solubility and absorption and hence is used in medicine. Deficiency of this vitamin causes a lowering of the prothrombin value of the blood. Clinically, it is used in hypo-prothrombinemia *i.e.* in hæmorrhagic diseases of the new born, after surgical treatment of obstructive jaundice etc.

DIAGNOSIS AND TREATMENT:—Clinical medicine is not able to keep pace with the spectacular advances, constantly being made in vitamin research. We know now of a larger number of vitamins than of known deficiency syndromes. Avitaminosis is generally due to multiple deficiencies and the results of controlled experiments in animals by the deprivation of one single vitamin, cannot be taken as criteria for treatment in human beings. The causes of vitamin deficiency are either: (1) *primary* or (2) *conditioned*.

*Primary vitamin deficiency* is due to failure to ingest sufficient amounts to meet the body requirements. Factors responsible are, poverty, ignorance, improper cooking and self-imposed restrictions,

such as what faddists and eccentrics advocate and what people with a low standard of living have to follow; the high cost of living witnessed in the present day, adds to the difficulties very much.

*Conditioned deficiency* is due to: (1) interference with ingestion. (2) interference with the absorption; (3) interference with utilisation; (4) increased bodily requirements; (5) increased excretion; and (6) increased destruction.

*Interference with ingestion* is manifest in (1) anorexia due to alcohol, surgical operations, debilitating disease, congestive heart failure and pregnancy; (2) gastro-intestinal disorders like gastro-enteritis, vomiting, peptic ulcer, gall-bladder disease, obstructive lesions of gastro-intestinal tract; (3) neuro-psychiatric disorders such as psychosis, neurosis, migraine and anorexia nervosa; (4) unsatisfactory denture, edentulosis and loss of teeth; (5) therapeutic restrictions as in peptic ulcer, obesity, nephritis, allergy and diabetes; (6) monotony and lack of variation in the diet, especially when people have to depend on what is readily available in the market rather than on what they would like to have; and (7) disproportion in the protein, fat and carbohydrate ratios.

*Factors interfering with absorption* are: (1) Achlorhydria which interferes with the absorption of ascorbic acid; (2) sprue syndrome with its hypermotility and rapid passage; (3) gastro-intestinal disorders such as hypermotility, reduction of absorbing surface, loss of mucosal integrity as in cancer, intestinal tuberculosis and gastro-intestinal fistula; (4) biliary disorders, especially obstructive jaundice; and (5) therapeutic measures. The habitual taking of liquid paraffin or other allied preparations for bowel regulation has been shown to interfere with the absorption of vitamins. Aluminium hydroxide and kaolin absorb water-soluble vitamins from the gut. Resections and short-circuiting operations and fistulæ of the gastro-intestinal tract are known to produce macrocytic anæmia and the sprue syndrome. Severe catharsis also produces deficient absorption.

*Interference with utilisation* may be due to: (1) Hepatic dysfunction, *e.g.*, cirrhosis of the liver, chronic hepatitis and alcoholism. Carotene has to be converted into vitamin A; thiamine phosphoxylated into co-carbo-oxylase; nicotinic acid into co-enzymes 1 and 2. These changes may occur in different tissues but the bulk of evidence points to the liver as being the main seat of such changes. So in the case of dysfunction of the liver, not only the conversion but also the storage and release of these substances are interfered with; (2) hypothyroidism; and (3) malignant diseases of the gastro-intestinal tract.

*Increased body requirements* are due to: (1) increased activity; fever increases the basal metabolism by about 13% for each degree centigrade. Strong emotions and strenuous exercises also raise the metabolic requirements. Influence of sunshine in the production of

deficiency is well illustrated in the case of pellagra, where there is excretion of vitamin B in the sweat; (2) pregnancy and lactation. Nixon in 1942 found pregnancy neuritis and conditions allied to toxæmia and beriberi in undernourished pregnant women; (3) hyperthyroidism increases metabolism; (4) therapeutic measures like fever therapy, increase the metabolism also insulin shock therapy; and parenteral administration of large amounts of glucose increases the demand for co-enzymes. The water soluble vitamin B group especially thiamine, may be easily washed out of the tissues and used up by repeated glucose infusions resulting in water-logging which is a manifestation of thiamine deficiency. The most-important factor causing deficiency is the treatment with large doses of a single pure vitamin. Spies, Bean and Ashe (1939) and Syndenstricker (1940) found that symptoms of thiamine and riboflavin deficiency became more pronounced in pellagra after nicotinic acid treatment. Lehmann and Neilser describe signs of pellagra after large parenteral doses of thiamine. Bichol and Meulengracht (1941) reported the development of pellagra after treatment with riboflavin. Leitner (1945) describes signs of riboflavin deficiency after large parenteral doses of thiamine. Marion Richards (1945) produced fits in rats kept on pyridoxin-poor diet after large doses of thiamine, while fits could be prevented if small doses of pyridoxin were given before the thiamine. Farer (1947) found that administration of large doses of thiamin in inadequately nourished cases of pernicious anæmia aggravated the glossitis and caused diarrhœa and pellagral dermatitis, but no pellagra-provoking effect of thiamin could be found in the same patients, when adequate diet was given. He also found that hæmatological relapse appeared quicker when nicotinic acid was administered after stopping liver treatment. Davidson and Girdwood (1946) observed in two cases of pernicious anæmia and three cases of sprue, during treatment with folic acid of vitamin B-complex deficiency *i.e.*, peripheral neuritis, cord involvement, angular stomatitis, *cheilosis* and pellagrous dermatitis. Whilst administration of purified vitamins was of no avail, potent liver extracts always brought about improvement. So it is an established fact that the administration of large doses of one pure vitamin easily upsets the balance in deficiency states, and exerts an inhibitory effect on other enzyme groups which are present in abnormally low amounts at the particular time. The well-balanced multiple enzyme systems present in potent liver extracts may restore the disturbed equilibrium even if they do not contain large doses of any of the known purified vitamins. The usual practice of injecting vitamin $B_1$ in massive doses may bring relief for the time being but will later entail more trouble to the patient.

*Increased excretion* may take place in: (1) lactation; (2) polyuria or forced water-intake, which may cause increased excretion of vitamins *e.g.*, urinary infections and sulphanilamide treatment; and

(3) excessive sweating which produces a deficiency of the water-soluble vitamin B.

*Increased destruction:*—By feeding animals with raw egg-white a generalised scaly dermatitis, pruritus, spastic gait etc. can be produced. This is due to the glycoprotein contained in the raw egg-white combining with the biotin in the gastro-intestinal tract causing a biotin deficiency. Another deficiency is noticed as chastek paralysis in silver foxes fed on *raw* fish which destroys the thiamine but which can be prevented by *cooking* the fish. Factors leading to increased destruction are: (1) achlorhydria; (2) alkalies and sulphanilamides; (3) toxic doses of lead, arsenic and gold.

The early diagnosis of vitamin deficiency is exceedingly difficult. The early symptoms are often non-specific and laboratory methods are often of little help. The deficiency should be diagnosed before the symptoms have developed into the text book description of deficiency diseases. The tendency to attribute definite symptoms to a particular vitamin has no clinical bearing and experience shows that early deficiency takes the form of border-line states of ill-health owing to the multiple deficiencies. As manifold deficiency symptoms usually occur simultaneously, a wide variety of vague and interwoven symptoms is met with. The earliest symptoms consist of anorexia, fatigue, loss of weight, restlessness, irritability, parasthesias, cramps in the muscles, burning in the eyes, photophobia, dimness of vision and impairment of memory. In vitamin A deficiency, clinical signs are detected only in moderately advanced cases. The earliest change is xerosis conjunctiva, the lustre being lost if the eye is kept open for a few minutes. Burning of the eyes, lacrimation, photophobia and discharge are additional early symptoms.

Early changes of vitamin B deficiency are most frequently met with. Various members of the vitamin B complex are grouped together more by virtue of their association in nature than by any structural similarity. Hence deficiency symptoms of the various members of the group overlap one another. The earliest symptoms of thiamine deficiency are anorexia, fatigue, irritability, dizziness and apprehension. These symptoms are not specific and the laboratory methods available are not sensitive enough to determine their origin. But it has been found that these symptoms, if untreated, lead to more definite and pronounced symptoms of deficiency. The symptoms of early riboflavin deficiency are fatigue, loss of weight, inability to concentrate, nervousness, transient digestive disturbances, soreness of the corners of the mouth, burning pains in the eyes with visual disturbances, headache, dizziness, insomnia and depression. Paleness of the lips and angles are also early symptoms. Nicotinic acid deficiency is characterised by loss of appetite, loss of weight, general muscular weakness, lassitude, insomnia, vertigo, numbness, palpitation, flight of ideas, anxiety and confusion. This may be diagnosed as malingering or neurasthenia and valuable time may be lost in diagnosis.

Early vitamin C deficiency can be detected if there is a congested, swollen, purple red, easily bleeding gum. There is fatigue, weakness and also a moderate degree of anæmia.

Early vitamin deficiency cannot be detected by the ordinary laboratory tests. The blood picture may or may not show a moderate, microcytic or normocytic anæmia; blood sedimentation rate, total serum proteins, albumin-globulin ratio are all usually normal. The F.T.M. may show achlorhydria but this is neither pathognomonic nor characteristic; œsophagoscopy and gastroscopy reveal the same changes as are found in the buccal cavity. But the deficiency pattern of the small intestine found on X-ray examination with abnormal segmentation, pooling, disappearance of the herring-bone structure, obliteration and coarsening of the mucosal folds is indicative of a vitamin B-complex deficiency. Chemical estimation of vitamin A in plasma and the estimation of the dark adaptation tests are useful in the diagnosis of vitamin A deficiency. The determination and excretion of vitamin $B_1$ and $B_2$, saturation tests for the assessment of nicotinic and ascorbic acids are always of help.

DIFFERENTIAL DIAGNOSIS—Vague, ill-defined waxing and waning symptoms of early vitamin deficiency are also symptoms of neurasthenia. Weir Mitchell's treatment benefited some neurasthenics, but after careful study, one wonders whether most of them were not cases of early vitamin deficiency and whether seclusion and reassurance or proper feeding was the more important factor in their cure. This confusion between the two states led to the indiscriminate administration of vitamins but their administration without an underlying deficiency does not improve the patient. So, vitamins are administered as a therapeutic test and if there is quick disappearance of symptoms, the diagnosis is established of early vitamin deficiency.

TREATMENT:—The principle underlying treatment is to deal with the conditioning factors and supplement the tissue deficiencies. Food contains many unknown factors necessary for proper nutrition and these are not present in the synthesised vitamin preparations. *The basis of treatment rests on securing an adequate diet.* It must also be borne in mind that the provision of the average daily dose, is not sufficient to make up for past losses. Vitamins are accessory food-factors and must be administered orally. Parenteral administration may be restricted to emergencies and to those cases in which there is obvious interference with absorption. In early vitamin deficiencies five to ten times the minimum daily requirement may be given, *e.g.*, thiamine and riboflavin 10 to 15 mg., nicotinamide 100 to 200 mg., ascorbic acid, 150 to 300 mg., all in divided doses, three times a day. Fat-soluble vitamins must be given in large doses as 50,000 to 100,000 I.U. of vitamin A daily. Large parenteral doses of pure vitamins, especially of the B group, may provoke a serious imbalance and hence it is advisable to give instead of

the most obviously lacking factor, a mixture of thiamine, riboflavin nicotinic acid and folic acid with Brewer's yeast. The treatment of early vitamin deficiency is an individual medical problem requiring patient diagnosis and properly planned therapeutic measures.

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## *Effective and Economic Treatment of Acute Bacterial Pneumonia at Home*

In an attempt to develop a simple effective and inexpensive method of treatment of pneumonia which could be employed in the home McVay and Stern of the University of Tennessee College of Medicine made a preliminary study of blood levels using repeated small doses of aureomycin. They were satisfied that the adoption of such a therapeutic method for pneumonia offered definite advantages:—economy, broad antimicrobial effectiveness and minimal toxicity, and the great practical importance of its being capable of being employed in the home by untrained personnel.

74 cases of acute non-tuberculous bacterial pneumonia were treated by them with 250 mg. of oral aureomycin administered 4 times a day. 29 of these cases had proved pneumococcic pneumonia; the remaining 45 had pneumonitis caused by *str. pyogenes, micrococcus pyogenes* var. *aureus* and gram negative bacilli. Bacteræmia was present in 8 cases. One case of acute primary Friedlander's pneumonia with bacteræmia was included in this series. 23 patients were atleast 50 years of age and 9 were critically ill. Very good clinical results were obtained. Empyema occurred in two cases and sterile pleural effusion in 3 patients. There were no deaths at all. The therapeutic regimen followed in this study is well adapted to the home treatment of the average case of acute bacterial pneumonia.—(*Antibiotics and Chemotherapy*, April 1952, pp. 215–220).

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### *Aneurin (Thiamine Hydrochloride) and Intolerance to Opiates*

Morphine in therapeutic doses produces in certain susceptible persons, vomiting, headache, vertigo and even a state of shock. This susceptibility has been observed more often in women and children. Lonjumeau and Leveque of the Paris Cancer Hospital, found that the simultaneous injection of aneurin (100 mg.) with morphine in a large series of cases of carcinomatosis, completely prevented symptoms of intolerance. They suggest that morphine through its action on respiration and circulation and on hæmoglobin, adrenals and liver, sets up an acidosis which affects the nerve cells and requires additional thiamine hydrochloride for its reversal by the decarboxylation of pyruvic acid.—(*Bull. Acad. Nat. Med.*, Paris., 138 : 45–48, 1951).

# THE EPILEPSIES*

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WE start with an apology for the title of the paper which we deliberately call "the epilepsies"—as such epilepsy that we know of comprises a group of heterogenous disorders which are characterised by a tendency to epileptic fits that recur over long periods of time. The days are past when epilepsy was considered and described as a particular disease. Now with the development of our knowledge as regards the various ætiologies, it has been possible for us to differentiate a few epileptiform fits from that group which is still very large and whose ætiology is still shrouded in mystery. So, we now prefer to call it as "the epilepsies". In recent years distinct groups of cases such as pyknolepsy, cysticercosis epilepsy etc. have been separated from the common term of "the epilepsies". Such epileptiform seizures as Jacksonian epilepsy have a definite pathological condition and have been completely separated from the common epilepsy of unknown ætiology. In the same way narcolepsy was originally included as a form of the so-called epilepsy but is now separated from it.

We, therefore, consider that "the epilepsies" is a better expression to designate that group of epileptic disorders of unknown ætiology which is known as epilepsy, as with the extension of our knowledge, other disorders with distinct ætiology and prognosis may be isolated from the common pool of epilepsies. This is understandable when we consider the case of an elderly person who is very fit mentally and physically, but on rare occasions has a few fits for which no cause can be found, and is classified together with a confirmed epileptic who started the fits in childhood with regular aura and a deteriorating mental faculty. It is just possible that some day we may discover the cause of the first whose prognosis is perfectly good and has no relation whatsoever with the second. It comes to this then, that epilepsy as we now understand it, is made to include various conditions, due probably to different pathological processes and different ætiological causes, some of which at any rate are still beyond our knowledge. Hysteria may also be included in that list.

Epilepsy has become such a familiar term even amongst the lay public that any fit from whatever cause is considered epilesy. A doctor seldom has the opportunity of seeing an actual fit. So he asks if the patient has had an epileptic fit. The physician is conversant with the various manifestations of an epileptic fit, from its

* Specially contributed to THE ANTISEPTIC.

various aura, *grand mal* to *petit mal*. We are therefore, not concerned with the description of epileptic fits. The physician has to depend mostly on the history of the attack furnished by the patient's relatives and friends, and decide if it was true epilepsy or not. Fortunately epilepsy has such a clear-cut pattern that it is not usually difficult for the doctor to come to a definite conclusion from the account furnished to him.

In the case of nocturnal epilepsy which is not very uncommon, but which may not be observed by others, there may be unmistakable evidence of the attacks *e.g.*, subcutaneous effusions about the face and neck, the patient falling and injuring himself and losing consciousness, the tongue having been bitten, or urine being passed unconsciously. These are the points that will help the doctor in right diagnosis. If the patient feels bruised and sore in the body on rising in the morning and if he suffers from a frontal headache, a nocturnal epileptic fit may be suspected. Such suspicion will be confirmed if there is in addition, a story of bitten tongue or bed-wetting or both.

A case reported by Bramwell is of interest. "The patient, a man well beyond middle age who occupied a prominent position in the town in which he lived, had for more than twenty years been subject to occasional epileptic fits during sleep. He had received no treatment and even his intimate friends knew nothing of his trouble. It was pointed out to the patient and to his son, a medical man, that any therapeutic intervention might alter the time of occurrence of the attacks; but they decided to take the risk. Six weeks later the patient had a major fit, while walking in the street, the first he ever had during day."

There should not be much difficulty in differentiating hysteria from epilepsy. The modern girl, who under the stress and strain of the present-day civilized life, is physically and mentally much more active than her sister of thirty years ago, is not likely to have much scope for hysterical manifestations.

Then again there is the question of faints and fainting fits. These may closely resemble *petit mal*. In faints, some definite cause, such as weakness after long illness, a stuffy room, postural hypotension starvation etc., is often to be found. It is usually preceded by a feeling of faintness which is not like an aura. Again the consciousness is lost gradually in faints and lasts longer; and the return to consciousness is a more gradual process. After an epileptic fit of a minor variety the patient either works normally or may be in a state of post-epileptic automatism which is not the case in faints.

Attacks of loss of consciousness in an adult should always arouse a suspicion as to its true epileptic character. Such attacks of unconsciousness and convulsions in an adult may be due to a variety of causes, *e.g.*, chronic alcoholism, chronic lead poisoning, syphilis as in G.P.I., uræmia, eclampsia, injury to brain, cerebral vascular

catastrophe, Stokes-Adams syndrome, encephalitis, tetany of any origin, hypo-glycæmia, cerebral neoplasms and cysticercus cellulosæ infestation. But their differentiation from true eplilepsy is not very difficult excepting in one or two cases.

Recently emphasis has been laid on the value of electro-encephalogram in the diagnosis of epilepsy, and the conclusion has been reached that paroxysmal cerebral dysrhythmia appears to be the basis of idiopathic epilepsy. About 60 p.c. of the relatives of epileptics as compared with only 10 p. c. of normal persons without such family history, showed abnormal E.E.G. tracings. The abnormal brain wave which forms the basis of epilepsy is in itself genetically determined and possibly inherited as a dominant Mandelian characteristic. Not all of them suffer from epilepsy—probably about 4 p.c. may do. These abnormal waves indicate that the subjects have functional instability of the nervous system associated with a lowered threshold to convulsions from even trivial causes. Disordered behaviour in some psychopathic personalities also give similar brain wave tracings.

Recently however, Funkhouser of West Virginia, after an extensive use of E.E.G. came to the conclusion that we must abandon the traditional description of epilepsy as *grand mal* and *petit mal*. On the basis of a physiological consideration of the age and also of the E.E.G. findings he classified epilepsy into three groups :—

The first group relates to the convulsive disorders which include the classical *grand mal*. This type occurs at any age and is characterised in the E.E.G. by sharp, sudden deflections called spikes. He also found that those exhibiting these particular features in the E.E.G. usually benefit by dilantin or phenobarbital, but are not suitable for tridione therapy, which may actually aggravate the condition.

The second group consists of the *petit mal* types. This type of epilepsy occurs principally in children. Its characteristic E.E.G. shows alternating spikes and slow waves. This type is usually benefited by tridione and not by dilantin which generally aggravates the condition.

The third group of epilepsy occurs principally in young adults. It is manifested by focal electro-encephalographic abnormality or spikes in one or both temporal lobes and is seldom benefited by medicine and is frequently aggravated by phenobarbital.

The question of allied disorders—which are often classed as psychomotor epilepsy probably include, according to Funkhouser, many conditions which have been loosely termed as "epileptic psychosis", "epileptic fugue," "epileptic furor", "post-epileptic confusional state" and "epileptic automatism." Even more important than all these, he thinks, probably many cases of hysteria are also

included in it; as also somnambulism and other unclassified psychoses. We have said before that from the E.E.G. point of view, psychopathic personalities behave in a similar way. Similarly aggressive psychopaths are sometimes indistinguishable from those of idiopathtic epilepsy. Funkhouser considers that inexactness of neuropsychiatric nomenclature has confused the issue and has created a great error of diagnosis—obscuring the epileptic condition which may be the real basis of the symptoms.

This is interesting in view of what we said above *viz.*, new conditions with distinctive ætiologic and prognostic features are being diagnosed out of the common pool of epilepsy. In that sense the range of epilepsy is gradually narrowing down, while according to Funkhouser, it is ever widening by embracing many psychopaths and even many hysterics. It will not therefore, be unreasonable to expect a description of epileptic personality. Such a concept is within the range of probability. He says that the clinical picture is so definite that a good neuropsychiatrist should be able to make a diagnosis of epilepsy on the basis of the personality make-up without a knowledge of convulsions. In this connection we are tempted to quote an incident narrated by Bramwell. "Some years ago the senior assistant in one of the University departments asked the professor, a distinguished clinician, whether he would like to see a lad who was applying for the post of laboratory boy, before he was finally engaged. "Do you take fits?" was the Professor's first remark, and the reply was "No sir, I have never had a fit in my life." That afternoon the boy had a generalised convulsion. Whether the incident was to be regarded as an illustration of the Chief's remarkable diagnostic acumen, or whether, alternatively, the awe-inspiring interview had determined the attack, furnished a fertile topic of conversation among the junior member of the staff for many a day." Even on the basis of second alternative, the boy must have been a potential epileptic.

The physiognomy of the chronic epileptic is often characteristic. A peevish, sensitive, egocentric, impulsive, sadistic, antisocial patient with "plateau speech" and often physical stigmata was described. But the leading epileptologists to-day believe that what was described as epileptic personality was the result of institutional treatment and chronic drug intoxication particularly as a result of bromide medication. But Funkhouser will not agree to this. He says there is no justification for abandoning the belief in epileptic personality. His arguments are that if psychopaths and behaviour-problem-persons can, even in a few instances, be definitely shown, to be suffering from epileptic equivalents, there may be more hope for them than would exist otherwise. This brings us to the question of the relationship of these personalities to epilepsies. Funkhouser has raised the question whether there is any valid correlation between abnormal brain waves as traced by E.E.G. and psychopathic

behaviour, criminality, hysteria and other miscellaneous neuro-psychiatric conditions. He feels that through the muddled haze of various concepts, it seems to become more clear that certain psychopaths and hysterics, and a great many miscellaneous personality deviations that are now classified as epilepsy, do manifest abnormal E.E.G. waves which are similar and in some cases indistinguishable from the brain waves of epilepsies. If such conditions can be allied to epilepsy—then as epilespy is controllable, he feels a better prognosis await all of them. An operation on the anterior part of the temporal lobe in certain selected cases of psychomotor epilepsy has succeeded in producing at least a temporary remission of symptoms.

It will be interesting to note here that there has been speculation even among the Freudians as to the basic origin of epilepsy. According to them *petit mal* is a substitue for masturbation and the rhythmical *grand mal* fit, a symbolic representation of regression to intrauterine behaviour levels and of death and rebirth fantasies. It is not possible in a paper like this to deal with this aspect in any great detail. However the E.E.G. has effectively dispelled much of this speculation.

In this connection it must be borne in mind that although emphasis has been laid on psychomotor epilepsy as a separate and distinct type of seizure, it is not implied that it exists only in a pure form. Just as a patient may suffer from both *petit mal* seizures and convulsive seizures, so may a patient have both *grand mal* epilepsy and psychomotor seizures. Although a psychomotor epileptic is not a criminal by nature, he may frequently be involved in police cases because of his disease which often makes him appear to be drunk, or which leads him to senseless destruction or aggressive behaviour. Without an electro-encephalogram it is extremely difficult to distinguish such cases from hysteria, psychosis or malingering.

Such probabilities of criminal acts by an epileptic brings us to the question of post-epileptic automatism. Such post-epileptic mental disturbances may have important medico-legal bearings. Automatic phenomenon sometimes follows an attack of *petit mal*. The patient in such a state is confused and acts in a way apparently oblivious of his surroundings and the persons around him—and when he comes round he violently denies having done any such act. Sometimes as an immediate sequel to an attack of it, the patient may be violent, maniacal and even homicidal. From time to time an epileptic may be guilty of indecent behaviour, kleptomania and various other crimes. We have seen cases, when a person after an attack of epileptic fit, even committed murder and later pleaded total ignorance of having done such a crime, and there was no reason to disbelieve his statement. It is however, not quite clear whether such criminal acts are the direct result of post-epileptic automatism *i.e.*,

a sequel to a fit or whether they are independent of the fit, being the so-called epileptic equivalents.

Occasionally a medical man may be called upon to testify to the fact whether a person can have an epileptic fit without his knowing it. This question may arise particularly in relation to marriage and in connection with life insurance. Cases have been reported in which the Insurance Company refused to pay the amount due on the ground that the party insured did not disclose his being subject to epileptic fits in the proposal form. Again this point may arise in the matter of divorce or dissolution of marriage, if one party being subject to epilepsy kept it deliberately concealed from his or her *fiancee*. It may be that the person was unaware of the fact that he or she was a subject of epilepsy. A day-epileptic will certainly know about his epileptic fits. But it may just happen that nocturnal epilepsy may occur without the individual knowing anything about it.

Another big problem in epilepsy with which a medical man is intimately concerned is in relation to marriage. The sentiment which governs marriage is a personal question and the bringing forth of off-spring is also a personal and sociological problem. A doctor is consulted not so much to pronounce on the parties' sentiments but to give them a definite opinion as to the result of marriage on epileptic fits in and the fate of the offspring. A doctor, particularly if he happens to be the family physician, must tell the party not to withhold the fact that he or she is subject to epilepsy—so that much unhappiness and recriminations in their future marital life may be avoided.

The anxious parents or the parties themselves may ask a doctor when they contemplate marriage for detailed opinion of their epileptic child. The doctor is bound to tell them the actual facts and leave the question of marriage to their judgement. They may be told that marriage will probably have no influence one way or the other, upon the pregnancy or severity of the fits. As regards the future of the off-spring it is more a matter of chance. There is no evidence to show that the child of an epileptic will necessarily be an epileptic; he may be a potential epileptic. Considering the fact that the parents and about 60% amongst the relatives of an epileptic may show cerebral dysrhythmia—it is probable but not certain that the child of an epileptic may inherit that cerebral dysrhythmia as recorded by E.E.G.

With a predisposing factor such as inheritance of an instability of function in the cells of the cerebral cortex being present, contributory circumstances in the life of the individual may produce the clinical syndrome of epileptic seizures. To this extent it is a risk—although the hereditary nature of epilepsy is grossly exaggerated. With this information it is the parties who would decide if they would take that slender risk—as after all the feelings which induce

marriage are usually too strong to be amenable to the doctrine of probabilities.

Another problem that may sometimes tax the knowledge of a medical man is in relation to compensation in a factory worker. A man may fall down and receive some injury in the head and that trauma may determine the subsequent epileptic attacks. While admitting that it may be possible—one has to ask whether that was a Jacksonian type of epilepsy or not. In the case of idiopathic epilepsy one has to satisfy the question whether the trauma was the result or the cause of an epileptic fit. In the absence of any history or clear evidence of previous fits, one has to admit the trauma as the cause and it becomes stronger; the shorter the time-interval between the accident and the fit, the more probable is the relationship. In a court of law it is better to rely on the opinion of the authorities than to venture one's own opinion on such a controversial issue.

PROGNOSIS:—Convulsions in an infant or a child always alarm the parents. Under the circumstances their overwhelming thought is what will happen to the child. Such a question from the parents is a perfectly legitimate one and a physician is expected to give his opinion.

When epileptic convulsions occur in infancy or early childhood with a history or evidence of birth-injury or defective mental development it is of serious prognostic moment. On the other hand, the occurrence of convulsions at the time of teething or at the onset of febrile disorders in childhood cannot be said to indicate necessarily a predisposition to epilepsy in later life.

Again pyknolepsy which has not long ago been isolated from "the epilepsies" is met with in children between the ages of 3 and 12 and is characterised by very frequent fits which are indistinguishable from *petit mal* but unaccompanied by mental impairment, in marked contrast to the frequent attacks of minor epilepsy in children in whom progressive mental deterioration is so common; and this condition *viz.*, pyknolepsy is not influenced by any drug—as is the case in epilepsy—which is an important point in diagnosis. Such fits invariably cease about the age of puberty and do not recur.

Idiopathic epilepsy on the other hand may commence at any age, but in the great majority of cases, the first fit occurs in childhood reaching the maximum about the time of puberty.

Epilepsy commencing after twenty (and the probabilities increase as age advances) should always arouse suspicions of an organic background and a diligent search should be made to find the cause. Brain tumour is not an uncommon cause of epilepsy; general or local fits may precede by years both the manifestations of increased intracranial pressure such as headache, papilloedema etc., and focal signs indicative of local damage to the brain. General paralysis of the insane is another condition in which recurring epileptic fits may

occur at a time when the characteristic symptomatology of the disease is comparatively inconspicuous. Cerebral cysticercosis is another cause of epileptic fits in adults. The diagnosis may be established if cysts which are of the size of a pea or larger, are palpated in the subcutaneous tissues, while calcified cysts in the muscles may be demonstrated by the X-rays. Stokes-Adams syndrome which is due to heart block and which is associated with bradycardia must also be borne in mind. Other conditions which may be responsible for epileptic fits in adults are chronic interstitial nephritis, acute alcoholism and lead poisoning. The progress in such cases depend on the causal condition.

The anxiety of the parents who witness generalised convulsions of their child is understandable. To a layman the sight is an alarmingly distressing one. The question uppermost in their mind is the possibility of death during a fit of convulsions. A medical man can allay that fear—as a matter of fact, death during an epileptic fit is very rare, and the risk can be dismissed as negligible. On the other hand, the status epilepticus, in which fits recur in quick succession with no return to consciousness in between, is a grave condition which may even terminate fatally. It is said that epileptics occasionally die from asphyxia or smothering when a fit occurs during sleep, or from choking when the fit takes place while he is eating. But such accidents must be very rare. Drowning accidents even in shallow water, are sometimes responsible for death ; and so no epileptic should be allowed to bathe in a bath, pond or tank. Epileptics should never be allowed to drive a car or ride a horse. This restriction is of paramount importance not only for his own good but also for the safety of the public. Epileptics may not be engaged in hazardous occupations, where liability to accidents is common.

Apart from all these considerations the prognosis in the epilepsies is wrapped in uncertainty. Spontaneous arrest occurs in a small proportion of the idiopathic cases and treatment may effect a cure. When the attacks are infrequent and of the major variety, when there is no mental impairment and when the case is seen in its early stages, the prospect as regards arrest is more favourable than is otherwise the case. But the response to treatment provides the most useful criterion in this connection. When mental deterioration sets in; the prognosis becomes grave so far as the liability to recurrence of fits is concerned ; but even then, it is not prudent to give a bad prognosis to the patient or his relatives until treatment has been given a fair trial.

PRINCIPLES OF TREATMENT :—Until the discovery of the new drugs, bromides and luminal formed the sheet anchor in the treatment of epilepsy. These should be taken with the utmost regularity and for a considerable period.

Luminal is often preferred to the bromides, since it does not produce a depressing effect—although the latter may be more

effective in some cases. If it is decided to use luminal, it should be given in doses of 1 to 1½ grains once, twice or thrice a day, depending upon the time when the fits occur and on the results obtained. Sometimes luminal produces an itching erythema which may pass off. But if it persists, the substitution of luminal sodium, the soluble salt will be found advantageous. Very rarely luminal may cause slight diarrhœa; and the dose recommended may sometimes produce undue drowsiness. The aim of the treatment must be to find the minimum dose which induces the maximum effect. Those authorities who have had extensive experience with this drug assert that luminal continued for many years has no untoward effect. We have seen a patient who used it every night for over six years without any ill effect, and he took it for insomnia. In the treatment of epilepsy with luminal the dose must eventually, however, be very gradually reduced, for the sudden cessation of luminal may precipitate status epilepticus.

Bromide is the drug of choice if given with due caution. It can be given alone or together with luminal. The maximum dose need not be increased beyond 60 grains in a day. One minim of Fowler's solution should be added to every ten grains of bromide as this will almost certainly prevent the occurrence of bromide rash. Any bromide may be used although some prefer the sodium salt; but there is no evidence to show that one is more efficacious than another. Sometimes it is observed that the effect of bromide or luminal becomes more striking if it is combined with either borax, zinc, belladonna or cannabis indica. It is a good plan when prescribing bromide to add not only Fowler's solution but also borax and/or Tr. Belladonna for long continued use.

Amongst the newer drugs sodium diphenyl hydantoinate (Epanutin, Solantoin, Dilantin) in a dose of 1½ grains twice or thrice a day, either alone or preferably with phenobarbitone has a good effect. But toxic symptoms may follow the administration of this drug and these are ataxic movement, dizziness, tremor, nausea and even diplopia; prolonged use may also lead to hyperplasia of the gums.

In case it is decided to substitute Dilantin or Epanutin for phenobarbitone it should be done gradually by reducing the dosage over at least a fortnight—for the same reason as mentioned above—as a sudden withdrawal may lead to status epilepticus.

A still newer compound tridione has been favourably reported upon in cases of minor epilepsy in which the E.E.G. tracings show a spike and wave formation during the attacks. This drug is available in 5 grain capsules and up to four capsules a day can be given to an adult. It is a toxic substance and its use is not without danger. It may produce agranulocytosis, and when administered, a white cell count must be done at frequent intervals. The time of administration of these drugs is determined by the time of

occurrence of the fits *e.g.* for nocturnal epilepsy the main and the bigger dose should he at night and a smaller dose in the morning.

In this connection Funkhouser's method of choosing the appropriate drugs by the nature of the E.E.G. waves where facilities as mentioned before exist, should be borne in mind.

*Status epilepticus* is an alarming condition to be vigorously treated by all available means. It is best dealt with by giving 4 to 6 drachms of paraldehyde per rectum or three grains of soluble phenobarbitone hypodermically. These doses may have to be repeated and some cases need as much as 30 grains of bromide introduced through a nasal tube and repeated once or twice at hourly or two hourly intervals. An enema should be given and cardiac stimulants prescribed in case of collapse.

The general health must be attended to. A simple digestible diet and regulation of the bowels are other matters requiring the physician's attention. Alcohol should be prohibited. A ketogenic diet has not been found to be effective as a method of treatment particularly in adults. A well regulated routine life is of great importance.

Lastly, the question of when to stop treatment when the fits are arrested is a moot one. There is no general agreement as to the period that must elapse before one can speak of recovery. Some eminent authorities consider a three years' freedom from attacks as a proof of cure. But cases are on record in which relapses *did* occur when treatment was discontinued even after this period. It is perhaps advisable in such favourable cases to reduce gradually the dose of the drug after three years. Further it is prudent for such patients to take one grain of luminal every night with the greatest regularity for a number of years thereafter.

Finally institutional treatment in a mental hospital may be indicated and is imperative, where facility exists, in those cases in which dangerous attacks of maniacal excitement occur as a sequel to fits. There is no place for surgery so far as our present knowledge goes—except in focal epilepsy, where a definite organic removable lesion in the brain is detected. Some surgeons however, claim that an operation on the anterior temporal lobe in certain selected cases of psychomotor epilepsy has succeeded in producing at least 'a temporary remission of symptoms.

Psychotherapy has its place so far as it is directed against feelings of inferiority complex and self-consciousness as well as against other difficulties from which such a child may suffer. Children should be kept in school and adults encouraged to continue their profession and do their routine work. The other members in the family must be taught to adopt a reasonable and sympathetic attitude towards the patient. Over-protection, over-anxiety and over-solicitude for the patient should be discouraged and emphasis placed on preventing invalidism with too much self-consciousness.

# ARTIFICIAL INSEMINATION—ITS MEDICO-LEGAL AND SOCIAL ASPECTS*

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Introduction.—At a time when people at the helm of affairs and the WHO are planning means and measures for effecting a reduction in the phenomenal day-to-day increase in population, particularly in the Asian countries, it may sound odd that I should address you this evening on "Artificial insemination—its medico-legal and social aspects." Imagine for a moment the absolute misery of some of those barren couples who, for no known fault of theirs are denied the pleasures of parenthood. It is but humane that scientists have put into practice artificial insemination in Europe, U.S.A. and Canada chiefly to comfort the female partner of the barren couple. Recourse to this method for begetting children with the assistance of doctors naturally brought in several unforeseen legal and social complications.

The constitution of our country has liberally conferred on us equality and freedom and the fundamental rights guarantee several privileges to both man and woman alike. Advances in science, political reforms and even fashions from foreign countries are sometimes adopted by us. All things human are bound to change and this method which is the subject of intense controversy in the West, may come to be adopted also by the miserable barren wives in our country.

What is artificial insemination?—Artificial insemination is the introduction of semen into the vagina or cervix by a suitable appliance without sexual intercourse. This procedure is resorted to when there is absolutely no chance of begetting a child through co-habitation. We are aware of the fact that the primary object of marriage is the perpetuation of the human race and the Hindus do believe in the saying "అపుత్రస్యగతిర్నాస్తి" One in ten married couples is reported to be sterile and it is revealing that in 50% of these sterile marriages, the male partner is incurably and absolutely sterile. The male partner may be impotent. He may be potent but suffer from azoospermia. Deformed or nonmotile spermatozoa and/or want of liquefying material in the semen may account in some cases for sterility in the male. In congenital malformations of the penis such as hypospadias, the ejaculation falls short of the female genital tract. All that need be done in these cases is to deposit the semen by a suitable appliance into the genital tract of the female partner and this procedure is known as artificial insemination by the husband (A.I.H.) This procedure is devoid of any socio-legal problems. On the other hand if the husband is incurably sterile, a donor's sperm cells have to be deposited, and this is designated artificial insemination by a donor (A.I.D.); and this creates medico-legal and

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social problems of a controversial nature. A.I.D. is occasionally resorted to, for overcoming Rh incompatibility in a couple, as children born to them are either dead at birth or live only for a short time or continue to live as mentally defective children.

Historical.—Artificial insemination was known to Arabs in the early part of the 14th century, when an Arab chieftain jealous of the enemy's prized stallion, got enemy mares inseminated stealthily with the semen of scrub stallions. With the idea of high breeding of cattle Abbe Lazzaro Spallanzani, an Italian Professor, successfully inseminated in 1780 a female animal artificially. In 1907 the Russian Scientist Iwanoff widely practised this method on cattle and sheep. Artificial insemination has come to stay as the method of choice in high breeding of cattle in all modern farms in U.S.A., Europe and even in India. Many farmers take advantage of this, as no stud fees need be paid. Only a small drop, amounting to 0·01 c.c. of semen is necessary to inseminate the female. In 1940, more than a thousand cows in Russia were inseminated from one bull. Advantage is taken also of the fact that the semen could be transported by air as the sperms remain viable for 7 days if properly stored.

Authentic record of how artificial insemination was done successfully for the first time in the humans, dates back to 1799 when under the advice of John Hunter, the wife of a Strand cloth merchant collected with a suitable syringe the semen from her husband and injected it herself into her vagina as her husband was handicapped by hypospadias. Soon after World War I, several reports of A.I.D. appeared from France. Since then, several hundred cases were reported from England and 10,000 cases from one group of doctors alone in U.S.A. by 1941. Reports of artificial insemination of the wives of soldiers who were away in the front-line during the last War are not news to us. In these cases, the semen was despatched by air and great care was exercised to avoid interchange of specimens during transport. The percentage of success by A.I.D. is however, variable.

The selection of a suitable donor.—The donor chosen should be free from communicable diseases and also be of similar race, religion, build, colour, temperament and mental capacity as the husband. He should transmit no inheritable disease and preferably be of the same blood-group as the father, except when chosen to overcome Rh-incompatibility. This is to safeguard any future blackmail or fraud and to prevent the donor from claiming the child on the basis of blood-group tests. The likeness in the child may not be guaranteed and the doctor has to take every care in the selection of a suitable donor. Some are of opinion that the donor should be of mature age (30 to 40 years) and better that he is the parent of a couple of legitimate children. It is possible to assess in this age-group the character and viability of the individual. Others feel that young unmarried adult donors of sound mind are best suited as the

artificially created child would not then have legitimate half-brothers and sisters. Intern doctors are often chosen as donors in U.S.A. Each donor receives a fee of 5 dollars for the contribution. Abundant precautions are taken to prevent the possibility of the donor meeting the woman receiving the semen or even to guess each other's identity. The prospective parent will never be aware of the identity of the donor. Secrecy is absolutely essential as otherwise, the donor may blackmail and claim the child. Pooling of semen, or two to three inseminations in a month may be done so that the doctor himself may not know who the father of the child is. The consent in writing of the couple to A.I.D. is obtained in the presence of the doctor. No more records are maintained.

Points in favour of A.I.D.—A woman who is married, has a home and the resulting stimulus to motherhood, but who is frustrated by the sterility of her husband is in a different position from her unmarried sister. The inborn instinct of a woman is the desire to have children. She is in despair and on the verge of divorce and wants a child. Suitable children for adoption are not available and the woman would rather like to have her own child instead of having to adopt and the husband may like to see features of his love in the offspring than the adopted one and so the law should not be hard on them. Some women yearn for actual maternity and nothing less will satisfy them. No stigma is attached to such children for neither of the parents would leak out the information. Women have requested doctors to carry out A.I.D. in secret, so that it may save their husbands' self-esteem. On the other hand, the wife may be so desperate that the request comes from the husband who wishes that the doctor would "for God's sake do something for my wife". A man who loves his wife cannot but feel that her maternity is frustrated by him and rarely objects to A.I.D. if his wife desires it. Previous venereal disease may account for sterility, and the guilty husband is then prepared to make suitable amends to his wife. Eloquent testimony of the married couple is found in their great joy when the new boy (by A.I.D.) arrives. The barren but potent father continues his conjugal relations and identifies himself as the true father of the child and participates in the trials, tribulations and blessings of pregnancy, parturition and parenthood. Nothing prevents a woman of low morals, from resorting to other questionable means to beget children without the husband being aware of it. On the whole therefore, A.I.D. is a humanitarian procedure and brings happiness to the couple in rearing up a child.

Objections to A.I.D.—Remarks made by neighbours that the A.I.D. child bears a close resemblance to the mother and nowhere approaches the father are intolerable to the husband. The child may be psychologically upset if later on, he is told that the he is the product of A.I.D. Secrecy is difficult to maintain in cases of A.I.D. practised to overcome Rh-incompatibility as the neighbours are often in the know of the previous marital history of the mother

and of the blood-group of the resultant A.I.D. child, which is necessarily different from that of the father.

To dub a woman barren, sterility must be proved to be absolute and incurable. Can we with confidence say that the methods adopted to assess infertility are infallible? The future may hold promise of something by way of cure, to these barren couples. Donation twice a week from a single person produces 400 A.I.D. children weekly and 20,000 annually and when the identity of child is kept secret there is the risk of incest creeping into society. There is always the danger of the husband guessing the donor.

Abortion induced without indication is criminal and so should be artificial creation and there is no reason why unmarried women should not have recourse to it. The fact that it will be a bastard child cannot be ignored.

Question of legitimacy.—Children born by A.I.D. are considered illegitimate and the parents often resort to perjury to overcome the difficulty. Although there was no physical contact with the female by the donor, it is tantamount to adultery. In New York and Virginia bills have been presented to the respective Senates for legitimising children by A.I.D., if the husband of the prospective mother has given his consent.

Does A.I.D. constitute adultery?—In 1949 the Archibishop of Canterbury in a debate in the House of Lords affirmed that A.I.D. *was* adultery.

In Oxford (Vs) Oxford, where the wife contended that the child was by A.I.D. and not through actual sexual intercourse, the judge opined that as she had voluntarily surrendered to another person for the enjoyment of the wife by one other than the husband, it amounted in law to adultery and was treated as such. Grandparents naturally wish to know whether the child is through A.I.D. or born to their son, especially when titles and property are to be inherited.

"In the Slingsby baby case of 1912, the younger brothers alleged that the child born to the couple, while they were on holiday in California was suppositious and denied the right of inheritance to the child".

The Rt. Hon'ble Henry Willink opined that A.I.D. is a highly controversial and difficult problem and that a woman has no right to delegate to a doctor the responsibility of making the choice. But many feel that the legal complications of legitimacy, adultery, and inheritance should be overcome by suitable legislation, if the couple desire A.I.D. and the doctor is convinced that the desire is deeply felt and shared by both parents and the donor is of opinion that he may solve the tragic human problem.

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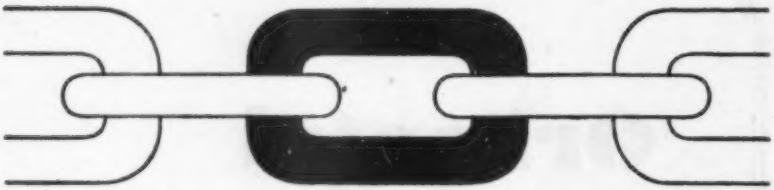

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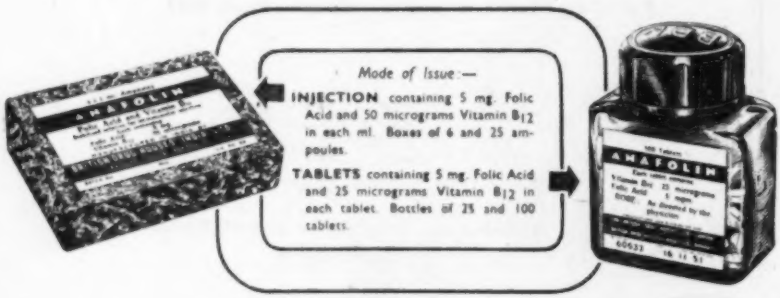
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# CŒLIAC DISEASE*

A. V. S. SARMA, M.B., B.S., D.C.H. (Lond.), F.D.S. (Lond.),
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Synonyms : "The Cœliac affection" (Gee) Acholia (Cheadle). Intestinal Infantilism (Herter).

CŒLIAC disease is a chronic disease of children who pass large pale offensive stools containing excess of split fat and in whom emaciation, arrest of growth and vitamin deficiencies develop.

Aetiology.—Hereditary influence if probably present. Recognised in childhood, the condition is said to be more common in girls.

Pathology.—The changes, *post-mortem*, are those of profound emaciation and due to complications.

There is a selective failure in the alimentary absorption of split fat, and even of glucose. Obstruction to lacteals, an insufficient secretion of bile, chronic intestinal infection (dysentery bacilli—Still), adreno-cortical deficiency, and defective pancreatic function have all been incriminated as causes of cœliac disease without adequate basis or positive proof.

The symptoms and complications of this disorder—anæmia, œdema, scurvy, tetany and rickets—are due either to impaired absorption or deficiencies due to restrictions in diet.

Wilfrid Sheldon (1948 and 1949) stressed intolerance to starches as of greater ætiological importance while Johnston and Howard (1949) suggest a hypothyroid constitutional basis; the above statements have therapeutic support.

SYMPTOMS.—The onset of the disease is gradual, between the 9th month and the end of the 2nd year. Loss of appetite, vomiting, failure to thrive, and diarrhoea appear, as also occasional febrile attacks.

In the well-developed state, the disease is characterised by very great emaciation which is so extreme that the child has the appearance of being merely skin and bone. The distended and tympanitic abdomen affords a striking contrast to the rest of the body. Loss of fat is obvious particularly over the buttocks, the tongue is clean, and the liver is small. Hypochromic microcytic anæmia is the rule ; but a megalocytic hyperchromic anæmia has been described in cases which persist into adult life.

The stools of an untreated case are large, unformed, soapy and offensive. The dried stool has more than 25 per cent of split fat, but the ratio between the unsplit and split portions remains unaltered.

* Specially contributed to THE ANTISEPTIC

COMPLICATIONS :—Oedema (ascribed by some to deficiency of vitamin B factors), purpura, scurvy, tetany and rickets are the complications in cœliac disease.

DIAGNOSIS :—Chronic indigestion, abdominal tuberculosis (particularly the ascitic type) and sprue (rare in childhood) are to be differentiated from cœliac disease. While the stunted growth is suggestive, it is the motion that is really diagnostic. The furred tongue, excess of mucus in the stools and enlarged liver are characteristic of chronic intestinal indigestion which is difficult of differentiation from cœliac disease, at least in the early stages. Intra-abdominal tuberculosis is diagnosed by palpating enlarged glands and by bacteriological methods.

COURSE AND PROGNOSIS :—A prolonged course and relapses are the rule in cœliac disease. Recovery occurs before puberty but cases may persist into adult life (non-tropical sprue). Growth is at a standstill during the active phases of the disease and also in cases where the disease has persisted into adult life (intestinal infantilism). Mortality is between 10 and 15 percent and is due to diarrhœa and exhaustion or some intercurrent diseases, like measles or broncho-pneumonia.

TREATMENT :—The crucial point is diet. High protein, low fat and starch-free diet is the aim. Skimmed milk, meat-broths and egg-white are ideal to start with. To these, dextrinised foods may be added. Banana, honey and sieved vegetables and a small quantity of butter are added. Multivitamin supplement containing, A, D, $B_1$ nicotinic acid and riboflavin, C and if necessary P and K is also allowed.

Dehydration consequent upon severe diarrhœa needs saline and glucose intravenously.

Skilled nursing, warmth, open air life and gentle massage are advised.

Bismuth preparations are used in diarrhœal bouts. Iron as Ferri *et* ammonium citras and crude liver extracts by intramuscular injection are needed ; the latter is now replaced by vitamin $B_{12}$ and folic acid. A boy of one year improved remarkably under liver therapy. Thyroid therapy is recently claimed as an useful adjuvant. Cœliac rickets needs vitamin D for prevention, during recovery.

*Illustrative cases* :—[*vide* Clinical photographs 1, 2, and 3 from "Cœliac Diseases," ANTISEPTIC, May 1944].

1. A boy, aged one year, admitted on 1-10-1943 for persistent diarrhœa of 1½ months' duration ; vegetarian and from a poor family.

*Family history* : nil particular.

*Clinical* :—An irritable and marasmic baby ; scabies present ; tongue and angles of mouth raw ; skin wrinkled and unhealthy and

in folds over buttocks. Abdomen soft and tympanitic; liver and spleen not palpable; heart and lungs: nil particular. Temperature is normal except for occasional febrile bouts, (about once in a month).

*Investigations*:—Kahn test of mother's blood: negative. Motion of the child: No ova or amœbæ seen.

Fæcal fat analysis (22-10-1943) under diet of albumin water, arrow root conjee and buttermilk:—Total fat 20%. Free fatty acid 12% and combined fatty acid as soaps 7·3% *i.e.*, 19·3% is split fat; neutral or unsplit fat being 0·7%.

Fæcal fat analysis (19-11-1943) with diet as before:—Total fat 24%. Free fatty acid 14·5% and combined fatty acid (soaps) 9·2% *i.e.* 23·7% is split fat. Neutral fat (unsplit) 0·31%.

Fæcal fat analysis (13-1-1944) with diet as before but with a little bread, diluted milk and tea flavoured with milk: Total fat 26%. Free fatty acid 10·8% and combined fatty acids (soaps) 14·1% *i.e.* 24·9% is split fat, neutral unsplit fat being 1·1%.

Culture of stools on 27-11-1943: No growth of acid-fast organisms in ordinary media (smear). No acid-fast organisms grown after 6 weeks' interval also. Culture on 24-1-1944: No growth of acid-fast organisms.

Blood (16-10-1943): Reds 3·3 millions; hæmoglobin 55%; colour index 0·83; whites total 10,500 per cmm; differentially whites are: P. 57%; L. 32%; M. 11%; E. and Masts 0%; Halometric reading (read) 7·8.

Blood (7-3-1944): Reds 4·5 millions; Hb. 70%; Whites 23,700 (patient has a few boils on the skin now). Differential count: P. 38%; L. 52%; M. 3%; E. 6·5%; Masts 0%; colour index 0·78; Halometric reading (read) 7·8.

X-rays chest (19-11-1943):—No prominent hilar shadows. Lung fields clear. X-rays abdomen: No area suggestive of calcified glands. X-rays of long bones: Marked rarefaction seen. Wrist: Dense white line of scurvy seen; (15-10-1943):—X-rays of tibiae, fibulae and wrist: Rarefaction still present with scorbutic dense white lines (3-1-1944).

Diet is albumen water, arrow root conjee, weak tea without milk, buttermilk (2 or 3 ounces per feed, at 3 hourly intervals).

1-10-1943. (1) Adexolin liquid 5 m., t.i.d. (2) Nicotinic acid 50 mgms. b.d. (3) Unguentum Danish for skin. (4) Calcium carb. gr. 30 mucilage acacia q.s. sugar gr. 30, Tr. catechu m. 30, Spts. chloroformi m. 10, Aqua cinnamomi ad 1 ounce; Fiat mistura; 2 drams thrice a day.

22-10-1943:—Same treatment with addition of pure vitamin C 25 mg. and calcium lactate gr. 30, per day allowed. From 1-12-'43

to 25-12-1943 lactic ferments were given orally. From 25-12-1943 liver extract was given on alternate days by the intramuscular route (12 injections of 1 c.c. each and later 2 c.c. twice a week : 12 injections).

*Progress* :

| Date | Weight | Clinical condition |
|---|---|---|
| 10-10-1943 | 9 lbs. | Several motions daily. |
| 17-10-1943 | ,, | ,, ,, |
| 25-11-1943 | 10 ,, | 3 motions per day. |
| 5-12-1943 | ,, | ,, ,, |
| 12-12-1943 | 11 ,, | ,, ,, |

*Progress* :

| Date | Weight | Clinical condition |
|---|---|---|
| 19-12-1943 | 13 lbs. | 3 motions per day. |
| 9- 1-1944 | 14 ,, | 1 or 2 motions per day. |
| 23- 1-1944 | 14 ,, | ,, ,, |
| 29- 1-1944 | 15 ,, | ,, ,, |
| 20- 2-1944 | 14 ,, | ,, ,, |

2. B.D. (clinical photograph) a boy of 3 years, admitted for diarrhœa and wasting. Blood : no parasite. Motion : no ova. Motion typical of cœliac disease. Treated as above with albumin water, arrow-root conjee, fruit juice, and with prolac. adexolin 3 drops twice a day. Marmite ¼ teaspoonful once a day. Weight on 27-4-1941 was 11 lbs. Weight on 4-5-1941 was 14 lbs.

Case 2. Cœliac disease.

Case 2. Cœliac disease.

Wasting and protuberant abdomen noteworthy.

The above cases are examples of cœliac disease. The clinical photographs are typical and speak for themselves.

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# THE RHESUS ANTIBODIES*

Lt. Col. G. W. G. BIRD,
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THE original work on isoimmunisation to the Rh antigens recognised antibodies active against the corresponding red cells suspended in saline. There was poor correlation however, between the clinical effects of isoimmunisation and the amount of antibody produced.

Race (1944) and Weiner (1944) almost simultaneously described a different type of Rh antibody which was called 'incomplete antibody' by the former, and 'blocking antibody' by the latter. This antibody united with the red cell antigens and coated the red cell surface, but agglutination of the cells failed to result *in vitro*. This type of antibody is the one concerned in the production of the clinical effects of isoimmunisation. Its presence can be detected either by indirect means (Weiner 1944), or by the ingenious test of Coombs *et al* (1945). Weiner's blocking test is based on the fact that when blocking antibody has coated the red cell surface, such coated cells will not be agglutinated by the ordinary antibody. The Coombs' test is capable of detecting antibody absorbed on to the red cells by different means. The red cells which are suspected of having absorbed antibody, are washed three or four times and resuspended in saline. Rabbit anti-human globin serum is then added and cells with absorbed antibody are agglutinated quickly and strongly on a tile or slide. This is because the absorbed antibody consists of human globulin. Diamond and Denton (1945) showed that these antibodies acted on the corresponding red cells when they were suspended in albumin. They called these albumin antibodies. They do not act on red cells suspended in saline. It should be noted that antibodies active against saline suspensions of red cells are also active against albumin suspensions. The term albumin antibodies refers to those active against red cells suspended in albumin.

Hill *et al* (1949-a) 'using a battery of serologic tests' postulated a third order of Rh antibodies. This type is incapable of agglutinating saline suspensions of the corresponding red cells, is incapable of 'blocking', may or may not agglutinate albumin suspensions, but gives a positive Coombs' test, and a positive hæmolytic test which had been previously described by Hill and his co-workers. The latter is much too difficult for routine work.

These authors use the term agglutinin to designate antibodies acting on a saline suspension of red cells, agglutinoid for albumin or 'blocking' antibodies, and cryptagglutinoid for those which neither 'block' nor agglutinate saline suspension of red cells.

* Specially contributed to THE ANTISEPTIC.

Confirmation of this concept was forthcoming from physical studies. Fractionation and electrophoresis revealed that agglutinins were limited to the *gamma* globulin fraction, agglutinoid was located in the euglobulin-albumin fraction, and cryptagglutinoid divided between the *beta* globulin and the euglobulin-albumin fraction. Further confirmation was obtained by chemical analysis of the fractions and a study of the effects of physical and chemical agencies on them (Hill *et al*, 1949-b) *e.g.*, heating to 70°C for one hour inactivates agglutinin but not agglutinoid or cryptagglutinoid.

A tabular summary of the types of Rh antibodies, synonyms, and their interactions is given below (modified from Hill *et al*).

| Antibodies | Synonyms | Agglutination, with saline suspensions of red cells | 'Blocking' test | Agglutination, with albumin suspension of red cells | Coomb's test | Hemolysis test |
|---|---|---|---|---|---|---|
| Agglutinins. | Complete agglutinins.<br>Saline agglutinins.<br>Bivalent antibodies. | + | — | + | + | + |
| Agglutinoids. | Incomplete agglutinins.<br>Blocking antibodies.<br>Albumin agglutinins.<br>Glutinins.<br>Univalent antibodies.<br>Hyperimmune antibodies. | — | + | +<br>or<br>— | + | + |
| Crypt agglutinins. | Incomplete agglutinins.<br>Blocking antibodies.<br>Albumin agglutinins.<br>Glutinins.<br>Univalent antibodies.<br>Hyperimmune antibodies. | — | — | +<br>or<br>— | + | + |

The various forms of antibody vary in their affinity for antigen in the probable order: agglutinoid, agglutinins, cryptagglutinoid. Antigen - antibody interactions however, follow the law of mass action and are greatly influenced by the concentration of the various forms of antibody present in a serum. Mohn and Witebsky (1948) have presented strong evidence for the presence of a fourth type of Rhesus antibody. It is likely that there may be many finer subdivisions, more of academic than of practical interest.

Summary.—Work is reviewed describing the present position with regard to the rhesus antibodies. At least three orders of rhesus antibodies exist. The types of antibody, their synonyms and various related tests have been summarised in tabular form.

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# THE MALARIA MENACE AND HOW TO AVERT IT*

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**PART IV**

THIS is the fourth and final article of the series on the malaria menace, published in the issues of the ANTISEPTIC for July '50, March '51 and Sept. '51 respectively. This article discusses further antimalarial measures as also the administrative aspect of malariology.

Bionomics.—Anophelene mosquitoes differ considerably in their bionomics and present problems necessitating modifications in control measures to suit local conditions. No hard and fast rules can therefore, be set down regarding the control of mosquito vectors :—

1. *Resting places* :—Immediately after emerging from the pupal acquatic stage, adults rest in places quite close to the breeding places. Later on, the females take to blood meal and utilize shelters in the vicinity of their hosts. During day time they rest in dark places with a low temperature and a relatively higher humidity than the outside atmosphere.

2. *Attraction and feeding habits* :—The biting times of various vectors have already been referred to in Article II. Only the females bite, the males cannot. "It is host-tropism more than susceptibility to infection which determines whether a local anopheline will transmit malaria." (Russel).

Ribbands states :—"Individuals differ in their attractiveness to unfed female anophelines, the attractiveness of one individual varies from time to time ; anophelines arrive at a hut to rest and then becoming hungry, seek food, not finding it, depart ; other anophelines arrive at a village attracted by the scent of the people and are not there by chance ; the number of anophelines attracted is proportional to the size of the community but the total number of anophelines is related to the capacity of the breeding places ; so the number of anophelines per person tends to be in inverse ratio to the size of the community and the malaria risk is greater in smaller communities ; this seems to indicate that under certain circumstances there would appear to be safety in numbers."

3. *Swarming and mating* :—Swarming or nuptial dance takes place immediately after hatching out ; they hover over a tall object, tree, shrub, building or even over the head of human beings for about 20 to 30 minutes, when mating takes place after a long and hard contest between the males. The female mates only once in its life and lays eggs after a blood meal. But A. stephensi has been observed to copulate repeatedly.

* The previous three articles of this series were published in the July 1950, March 1951 and September 1951 issues of the ANTISEPTIC respectively.

4. *Age*:—Longevity depends on climatic factors, it may be a few days, it may be a few months. Russel and Rao state that the "mean probable duration of life of an adult A. culicifacies is two days with a mortality rate of 50% every two days during at least one season of the year".

5. *Range of flight*:—The range for important vectors has already been described (*vide* ANTISEPTIC, 1951, p. 185). The range is usually not greater than a mile, but may even be 1½ miles, as in the case of A. culcifacies. Flight range of the local vector is thus a very important point in the planning of control measures.

6. *Attraction to light*:—Thompson found that mosquitoes leaving the shelter of the house at dusk or dawn were strongly attracted to light coming in through the window and door opening. To the restless mosquito, the attraction to light was equally strong during the hours of darkness, mosquitoes being attracted even to the faint light of the night sky. Reid states that:—(1) the mosquitoes are quiescent by day if not disturbed, whether kept in the light or in darkness; (2) if they are in complete darkness as at night or in a photographic dark room, they fly vertically upward; and (3) they are attracted by a dim light at night and will fly sideways or slightly downward, instead of vertically upward as in the dark room.

7. *Attractability*:—What Brown described for the Aedes, may be true also of the anopheles:—(*a*) moist clothing attracts 2 to 4 times as many as dry clothing, in temperatures over 60°F whereas the reverse is true below this temperature; (*b*) sweat, $CO_2$ and petrol attract them more; (*c*) white clothing is much less attractive than black; also light red or blue than dark; green was less attractive than either red or blue. Dark blue was slightly less attractive than dark red, whereas light blue was more attractive than light pink; and (*d*) satin was less attractive than broadcloth or crepe, and nylon much less attractive than cotton.

8. *Control of adult mosquitoes*:–Bats, clovers, planting barriers, deviating mosquitoes from man to animals, augmenting natural enemies (beetles, dragonflies), mechanical measures such as destruction of rank vegetation, swatting, hand collection, trapping, fumigation and producing smelly perspiration, are the various measures of control often suggested and applied but the most effective method of a mass attack on mosquitoes is the regular use of natural vegetable or/and synthetic insecticides.

*Fumigation*:—Pyrethrum, camphor, formic acid, stramonium, sulphur, datura, tobacco, chlorine, wood or straw smoke, cresol vapour have all been used as fumigants against mosquitoes in individual homes, but are of little value as community control measures.

*Direct kill*:—The spray-killing of mosquitoes with Ext. pyrethrum conc. solution in kerosene oil applied to buildings at a dose

of $^1/_2$ oz solution per 100 cft., has been found successful in Brazil, India, Netherlands and South Africa. This is also recommended by WHO in all cases where rapid disinfestation is desired as in treating aircraft, or in controlling a sharp and brief epidemic of malaria.

*Pyrethrum* (Chrysanthemum cinerariæ folium) is a plant which is grown chiefly in Dalmatia, Japan and Kenya, now also in South America, South India, Kashmir, Punjab hills, U.P., M.B. and Orissa. An effective spray can be prepared by merely soaking a pound of dried and crushed pyrethrum flowers in a gallon of kerosene oil for 72 hours (Covell). Emulsions have also been used but with less success :—

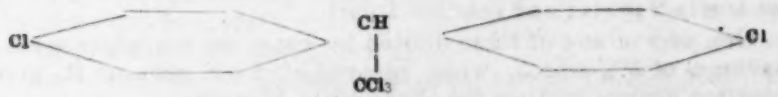
| | | |
|---|---|---|
| Pyrethrum flowers | ... | 20 lbs. |
| Sodium lauryl sulphate | ... | 184 gms. |
| Kerosene oil | ... | 12 gallons |
| Water | .. | 7 gallons |

This emulsion has an immediate knock-down effect but practically no residual action, and needs systematic, repeated, daily to weekly applications, according to the behaviour of the local vector anophelines. It is now used only in emergencies ; insecticides which have a permanent residual effect are now therefore, in routine use. *Derris* (Rotenone) is another natural insecticide used like pyrethrum with persistent residual effects.

Malaria control by residual insecticides is an extremely simple and direct method of eradicating malaria by interrupting transmission ; the spraying can be restricted to houses and shelters in malarious zones and need not be carried out in the whole area. By a change in the technique of spraying, malaria control of extensive rural areas has now become economically and technically possible. Of the various chemicals in this group DDT ; BHC ; DDD or TDE, Dieldrin, Toxaphene, Parathion, have all been tried ; also Chlordane, which according to Busvine has insecticidal powers which are intermediate, and which is similar to *gamma*-BHC as regards its volatility and toxicity to mammals.

DDT (dichlorodiphenyl trichlorethane) is the most widely employed insecticide at present in anti-adult-mosquito campaigns.

The biologically pure DDT has the following chemical composition :—2:2 BIS (P-Chlorophenyl) 1 : 1 : 1-Trichloroethane.




It is a white crystalline substance, with a melting point of 107°C to 108°C. It is stable and nonvolatile at atmospheric temperatures, unaffected by exposure to light, air, water, acids or alkalies, and is sparingly soluble in water, highly soluble in organic solvents and

crude oils, like fuel oil, cotton seed oil, castor oil, kerosene oil etc.; crude kerosene oil being the cheapest oil, solutions of DDT are made in it and used for house-spraying. DDT itself has no deleterious effect on fabrics or metals, but the organic solvents used may be harmful to paints, gold and silverware. It is primarily a contact-poison although it may also sometimes act as a stomach poison (Gupta, ANTISEPTIC, 1949, p. 852). Solvent oils after volatalisation, leave a fine film of the crystals of DDT on the sprayed surface, which acts as a contact-poison, through the tactile sensory organs in the tarsi of the insect, affecting the legs first and then spreading throughout the insect's body. The insect being thus rapidly paralysed by contact with the DDT is unable to feed or even to insert its proboscis into the skin.

Preparations of DDT usually applied.—*Solutions*:—A 5% solution prepared on a large scale by dissolving 8 oz. by weight of commercial DDT in one gallon of kerosene oil, is commonly used for indoor-spraying. The solution can be prepared by stirring or by melting the DDT in a tin and stirring it in the solvent or by heating the oil and stirring the DDT into it; the latter method being risky and liable to catch fire is best avoided.

*Emulsions*:—Different formulæ are used for emulsion concentrates:—In U.S.A., they use DDT 25 parts; Triton 10 parts; and Xylene 65 parts.

In India we use either:—Toluene 33·35; Turpentine 33·35; Abs: alcohol 4·4; Dry soap powder 2·8; Water 1·1; DDT 25·0; *or* DDT 50 gms.; M.K.E. 12 c.c., Oleoresin soap 20 gms.; Sodium silicate 3 gms.; Water 15 c.c.

This emulsion concentrate when mixed and stirred with enough water to give a 5% strength is used for indoor-spraying. Emulsion concentrates are less bulky to transport and are less toxic to operators when diluted. "An emulsion is considered satisfactory if, at a concentration of 1 per cent of DDT, no visible separation occurs within two hours and only moderate creaming with no separation of the oil-phase within 24 hours" (Jones).

*Suspensions*:—Water - dispersible suspensions are on sale as 'Geigy', and 'Neocid' powders covered by patent rights. Other useful formulæ are:—(1) DDT 30 parts; Gum acacia 1 part; *or* (2) DDT 60 parts; gelatine 1 part; *or* (3) DDT 120 parts; gum acacia 2 parts; and gelatine 1 part.

One part of any of these diluted in water to ten parts gives a suspension of 5% which, when sprayed at 2 c.c. per sq. ft. gives protection against malaria for about 10 to 12 weeks.

Powders of DDT or Gammexane P. 520 which can be wetted (12 oz. mixed in a gallon of water and applied at ½ gallon per 1000 sq. ft. so as to give 11 mg. *gamma* - isomer) have proved better for application in sprays to the mud walls of houses than solutions

or emulsions. Hadaway and Barlow state, "There is an inverse relation between the size of ground DDT crystals fractionated into size-ranges by sedimentation and their effectiveness. Smaller particles are more effective than larger ones, even when equivalant numbers are compared ".

The shape and mass of the crystals together influence, pick up and determine their effectiveness. "The order of toxicity when fresh, has been found to be BHC, chlordane, DDT and the order of the residual effectiveness has been DDT, BHC and chlordane" (Reid). BHC is found to have a marked repellant effect as compared with DDT etc.

*Paints*:—Campbell and West have recorded their experience of a DDT-oil-bound water-paint "which acts not only as a paint but has also an insecticidal action on flies (possibly therefore, also on mosquitoes) giving a 90% kill in 48 hours and having a residual effect". Whitfield has recently developed a cement-paint incorporating BHC or DDT, 30 minutes' exposure to which a week after painting, killed Aedes mosquitoes in 1 hour.

*Aerosol sprays*:—Sparklets and ærosol bombs combining DDT and pyrethrum have been put on the market from which vapour under pressure escapes and has an 'immediate knock-down effect on insects in an enclosed space. These are however, meant mainly for individual well-to-do homes.

*Aerosol dispensers* used for disinfestation and disinfection of air craft approved by the British Ministry of Health are:—(1) The Cooper DDT low pressure ærosol bomb containing pyrethrins 0·4% and DDT–3%; and (2) Cooper BHC. L.P. ærosol bomb containing pyrethrins 0·65%; and BHC—0·5%.

Methylene chloride and petroleum ether were tried as dispensers but abandoned because of the crazing effect of the former on plastics and of the inflammable nature of the latter; freon is recommended instead.

Spraying Technique.—Applied as a 5% solution, emulsion or suspension on walls and ceilings of houses at a dose of $^1/_2$ gallon per 1000 sq. ft., but one gallon in the case of suspensions, leaves a concentration of 100 mg. PP—DDT is lethal to mosquitoes and has a residual (persistent) effect lasting even for 10 to 12 weeks depending on the surface sprayed. Gammexane P. 520 (6·5 per cent *gamma*-isomer) 12 oz. in one gallon of water sprayed similarly has been found in the Belgian Congo to be effective for 15 or 16 weeks or longer. The dosage of $\frac{1}{2}$ gallon per 1000 sq. ft. gives a deposit of 11 mg. essentially 10 mg. *gamma*-BHC (*gamma*-isomer) per sq. ft. Gammexane liquid concentrate L.G. 140 containing 10% *gamma*-BHC diluted one to twenty volumes of kerosene and applied at $^1/_2$ gallon per 1000 sq. ft. (11mg. *gamma*-BHC per sq. ft.) has been

recommended in preference as it avoids the unpleasant odour produced by gammexane P. 520 aqueous solution sprays.

An uniform, non-trickling, homogeneous non-patchy spray in all buildings, huts and other constructions at two-monthly intervals has been found quite effective. Safe resting places for mosquitoes in houses such as, clothing, saddlery, untreated furniture, freshly spun cobwebs, and fireplaces, should receive special attention.

*Area spraying*:—Indoor spraying of buildings and hutments and the saturation of vegetation and all features within a radius of 50 yards around, are advised, particularly in aggregations of labour colonies etc. where more frequent and heavy doses are applied (once in a fortnight).

*Barrier spraying* advocated by Russel, Afridi and Nair is another method where a perimeter line of 50 yards wide around a labour camp is marked out and all constructions in this barrier area are sprayed ; thus preventing the infiltration of mosquitoes into the area under control. 2·5% DDT spray has been found to be effective but because of the larger dosage and greater frequency and time required, indoor spraying is found more economical.

*Air-craft spraying*:—The following recommendations are made by David and Busvine:—(1) An L.P. ærosol dispenser containing 0·4% pyrethrin and 3% DDT should be used in a dosage of 10 g. per 1000 cft. (2) A dispenser distributing not more than 1 g. per second, should alone be used as otherwise it will be difficult to cover the entire plane without overdosing. (3) The doors to be kept closed for not less than 5 minutes (or longer where possible) after complete spraying. (4) The spraying should be done after all the luggage has been loaded but before the passengers embark.

*Spraying equipment*:—Various kinds of sprayers have been tried but knap-sack sprayers (four oaks or Ross), and stirrup pumps are found to be efficient. The *stirrup pump* with a nozzle aperture of 1/32 inch, fitted with a relief valve to save hose-burst, has been in successful use in countries where labour is comparatively cheap. The knap-sack sprayer, with a nozzle aperture 1/32" (four oaks or Ross pattern,) requires one individual to operate, is more economical, and equally effective for suspensions as well as emulsions and is also a good and clean method. It is useful particularly where labour is costly and difficult to get, habitations are scattered and means of communication are not adequate or convenient. Where a truck is used, compressors for supplying air to the knap-sack sprayers have also been utilised. The knap-sack sprayer is therefore, the method of choice. A simple, improved hand-sprayer with a fan-shaped nozzle would be a great improvement, if one such can be improvised.

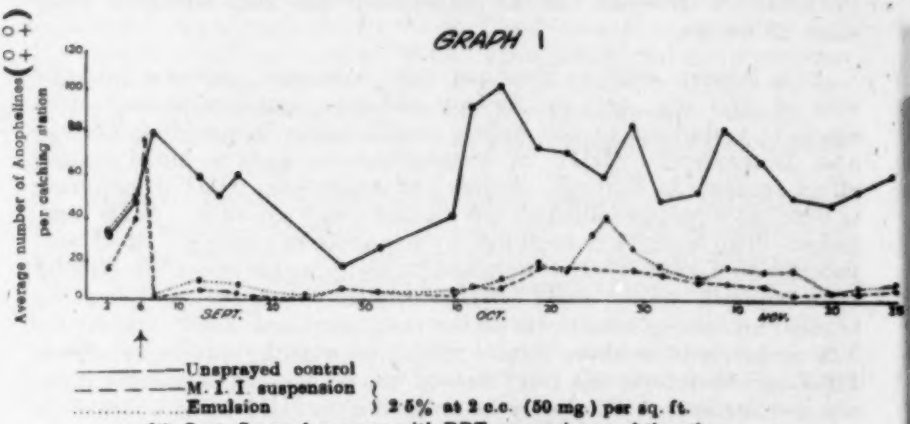
Residual toxic effects.—The effects of different preparations of DDT and BHC have been plotted out as Graphs I, and II, after

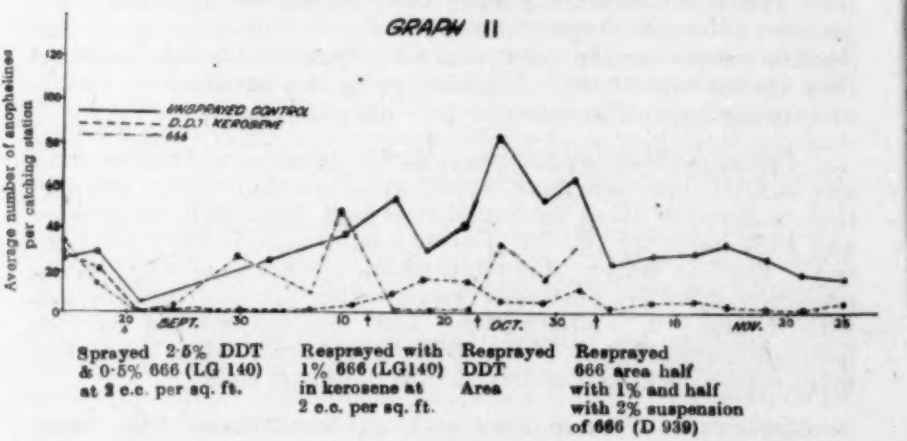
the pattern of Puri and Krishnaswamy. The behaviour of mosquitoes to pyrethrum, DDT and BHC can be summarised as follows :—

(1) *Pyrethrum* :—The knock-down is quick but no residual effect.

(2) *DDT* :—Knock-down slower, proportion killed within 24 hours lower than No. (1)

(3) *Gammexane* :—Knock-down similar to DDT ; proportion killed in 24 hours higher than DDT.


GRAPH I
Average number of Anophelines (♀♀) per catching station
SEPT.
OCT.
NOV.
Unsprayed control
M. I. I suspension
Emulsion
2·5% at 2 c.c. (50 mg) per sq. ft.
6th Sept. Sprayed one area with DDT suspension, and the other with emulsion (both 2·5% at 2 c.c. per sq. ft.)


GRAPH II
Average number of anophelines per catching station
UNSPRAYED CONTROL
D.D.T. KEROSENE
666
SEPT.
OCT.
NOV.
Sprayed 2·5% DDT & 0·5% 666 (LG 140) at 2 c.c. per sq. ft.
Resprayed with 1% 666 (LG140) in kerosene at 2 c.c. per sq. ft.
Resprayed DDT Area
Resprayed 666 area half with 1% and half with 2% suspension of 666 (D 939)

Wharton and Reed state DDT caused very little mortality, but had an irritant effect of driving the mosquitoes from the huts into the light trap and reducing the biting rate.

BHC caused a very high mortality in the period immediately following treatment, falling off gradually over the 12 weeks covered

by the observations. Like DDT, it reduced the biting rate, and to a greater extent, and drove the mosquitoes into the light trap." Cutkemp reports that "after 29 weeks, DDT was more effective than BHC or chlordane applied on beaver wall board but BHC acted more rapidly than DDT when deposits were not so old. DDT was less effective than BHC on a surface of red clay subsoil. The toxicity of chlordane on clay was very low after one week. All three compounds were much more effective on wall board than on clay; the action of DDT solution, emulsion and suspension was the same for 16 weeks but the suspension was still effective even after 29 weeks.

*On wooden walls*:—Five per cent kerosene solution (at the rate of 209 mg. per sq. metre) becomes completely ineffective against A. darlingi in the eighth month after application (Galvao and Damasceno). DDT in water-emulsion had a more lasting effect against A. darlingi. *Leaves and vegetation*: DDT in emulsion is best, as aqueous solutions are washed away by rain. *Glass surfaces*:—The toxicity is soon lost by exposure to sunlight. *Mud surfaces*:—Surfaces slightly roughened by scraping are more insecticidal than smooth ones. DDT—BHC aqueous suspensions leave the highest amount of insecticide on the rough surfaces. *Lime washing*:—The majority of workers believe that lime washing adversely affects DDT and therefore, the DDT should not be applied on freshly lime-washed surfaces. They may be treated with DDT after a lapse of 10 or 15 days. DDT may however, be applied mixed with distempers, prepared with a chalk base. *Other surfaces*:–Unpainted wood, plaster and cement retain the DDT for a long time, glass and window screens remain insecticidal for a considerable time provided they are not rubbed out. Old paint gives less satisfactory results and freshly painted surfaces are very unsatisfactory.

*Factors influencing toxicity*:—It has been found that the toxicity of DDT decreases more rapidly at a high temperature (98·6°F) than at low ones when the humidity is high. Both high temperature and high humidity (60–70%) cause a more rapid reduction in toxicity. Pradhan states, "the temperature coefficient of DDT entry, remains positive irrespective of concentration, but its value decreases with decrease in concentrations and becomes masked by the temperature coefficient of the insect's resistance when a negative temperature coefficient of DDT action begins to show".

*Resistance*:—Certain races of A. quadrimaculatus (Fay, Baker etc.), in some areas of Africa, Phillipines and a few other places are reported by the WHO Expert Committee on Malaria to have resisted the action of DDT. They also noted that DDT excitation or repellency was not confined to A. gambiae. Evidently gammexane was the answer. Other factors influencing efficacy are persons habitually sleeping outdoor, where they are exposed to the mosquito

bites and sprayed surfaces being subjected to frequent renovations or white-washings.

*Precautions*:—DDT or BHC as ordinarily applied for residual spraying is harmless to man and animals, but should be used with caution so as to avoid contamination of food and of cooking utensils. Containers should be properly and securely labelled. The whole operation should be in the hands of trained personnel.

Conclusion.—For the control and eradication of mosquitoes, DDT, and BHC are very useful being cheap; they have prolonged persistent action, are nontoxic to man and domestic animals, and may confer collateral public health benefits *e.g.*, the simultaneous destruction of other disease-carriers; but it would be unwise to discard the time-honoured land-reclamation measures of control in places where conditions are particularly favourable for anopheline mosquitoes flourishing.

*Measures directed against the malarial parasite*:—These consist mainly in the use of drugs (antimalarials) in the treatment of patients and carriers. A number of drugs (Quinine, Mepacrine, Chloroquin, Paludrine, Pamaquin, Pentaquin, Isopentaquin, Camaquin etc.) are in use.

In the control of malaria in any locality, this measure can play only a secondarly role. The WHO Expert Committee on Malaria, (third session) stated:—"While therapeutic and prophylactic antimalarial drugs should be available to those who require them (regardless of ability to pay for such treatment), it must be emphasised strongly that in numerous rural areas throughout the world the use of residual sprays has in two or three seasons made mass chemotherapy and mass chemo-prophylaxis of malaria unnecessary and obsolete. On the contrary intensive distribution of free antimalarial drugs for many years in other areas has had no noticeable marked influence on the incidence of malaria except under well supervised groups."

A considerable volume of work has been done in various countries on the use of antimalarials which are valuable in treatment; yet no drug is known to prevent a person from becoming infected with the parasite when he is bitten by an infected mosquito and no drug can be relied upon to destroy all stages of the parasite when once they have become lodged in the body. A drug to be considered specific should be effective as a causal prophylactic against all species of human malarial-parasites, and also be a good therapeutic agent and, at the same time, be truly curative; it should have a low toxicity and be readily available at moderate cost" (WHO).

Even if a drug answering all the above specifications could be found, the treatment of carriers would be difficult. This would involve blood examinations of the community as a whole, but the parasites although lodged in the body may not always be found

in the circulating blood (exoerythrocytic stages) the control of malaria by drugs in a malarious community, would imply that all persons whether showing parasites or not should be treated; this would be very expensive, and even impracticable while control by residual spraying and other antimosquito measures would be cheaper and more effective.

Other anti-malarial measures.—*Social measures*:—Malnutrition has no bearing on the incidence of malaria. The absence of sufficient public opinion regarding *(a)* the economic and public health importance of mosquito-conveyed disease; (*b*) the available measures of prophylaxis and control; (*c*) the social responsibility for preventive measures (Russel), is what requires to be remedied. Further a social, as well as an administrative measure is proper attention to the control of malaria in *tropical aggregations of labour*, the population of which is composed of elements forming artificial groups, subject to a regimen, which is different from the normal. The Malaria Commission of the League of Nations considered and experience has also confirmed that health must be preserved among these groups, irrespective of cost, as this is a useful investment in the long run. In such camps, appropriate measures of control must be selected depending on the size of the camp and its duration, the malaria season and the habits of the local carrier mosquitoes.

*Education and propaganda*:—Without the willing co-operation of the public and their realisation of the advantages that would accrue to them from the work of the technicians, no scheme designed to benefit the masses will succeed. Education and propaganda in malariology are therefore, essential. Public health propaganda is theoretically a simple matter but complex and difficult to put into practice. They should suit the needs which vary greatly according to race, religion, language and level of education etc. Health education in malaria must be imparted:—(*a*) to school children compulsorily, as is done in Venezuela; (*b*) to adults by means of cinema films, film strips, lantern slides, posters, pamphlets, exhibitions, show-stalls in fairs and festivals, and at public gatherings. Posters could be displayed at places where political or religious leaders address the public. Posters are the cheapest means by which to reach the maximum number of persons without involving elaborate arrangements or large expenditure; and (*c*) the WHO Expert Committee opines, 'that even more important than educating the general public is the vital need to acquaint administrative officers and engineers of all branches, with the basic principles of malariology. The aims of public education as enunciated by E. Rood (Malaria Education–Rural Press–Madison College, Tenn: 1939) are:—(1) To bring about a community understanding of the practical phases of malaria prevention and control, so that the action taken by individuals, by families and by communities may be on a concerted basis. (2) To develop ability in the individuals of a community to

protect themselves and their families against malaria. (3) To develop an appreciation of the seriousness of malaria as a communicable disease and to secure the voluntary co-operation of all citizens in its control and eradication. (4) To develop an appreciation of the need for adequate medical care for sufferers from this disease. (5) To develop a sense of social responsibility towards indigent victims of the disease, leading to some provision for their care and treatment. (6) To develop an appreciation of the efforts of the official health departments and to enlist hearty support from the members of the community.

With a view to educating the public, a Malaria Institute has been established at Delhi which, besides its numerous other functions, has "to provide instructional manuals on malaria surveys, entomology, anti-mosquito measures and other aspects of malaria investigation and control". A number of publications entitled Health Bulletins, and 'Malaria Series' have been published by the Government of India Publications Department. These are not only useful for administrative officers, health officers and engineers, but are designed to be of help to the educated lay public *e.g.*, 'Lectures on malaria,' 'Man-made malaria,' 'What malaria costs India'. These bulletins would prove valuable if included in the curriculum of medical studies.

*Administrative aspect*:—The food problem is linked up with engineering, food production and agriculture; a joint problem therefore, for two ministries. 'It is only by bringing under cultivation large areas now lying fallow, that the full requirements of the nation can be met. Many of these areas are so highly malarious that they cannot be brought under cultivation at present. If effective malaria-control measures are carried out, the food problem will also become easy. If a portion of the huge sums paid to other countries for foodstuffs is diverted to antimalaria campaigns, wonderful results can be achieved in a short time', The WHO malaria teams working in Teria (U.P.) and in Malnad areas have proved this by converting unhealthy malarial swamps into healthy valuable agricultural tracts.

*Quarantine*:—The expansion of international communications has created new malarial problems and the two most important dangers from air, sea or land communications which are envisaged by the WHO are:—(1) The spread of anophelines regarded as of major importance in the transmission of malaria to continents, or major subcontinental divisions outside their normal range of distribution, as for instance the spread of Anopheles gambiæ, culicifacies, punctulatus etc. (2) The redistribution of any acknowledged vector of malaria within the zoological area to which it naturally belongs, as for instance by the extension of its distribution to nearby countries or by the reinvasion of areas from which anopheline species have been eradicated.

It is with a view to meeting these two major requirements of malaria control, that quarantine and disinfestation measures are sought to be enforced on an international level. Thanks to the international conventions on 'yellow fever', this disease remains restricted to Africa and is not allowed to spread to any of the other countries where the vector mosquitoes (Aedes ægypti) flourish *e.g.* India. Rigid anti-mosquito sanitation, including the elimination or treatment of potential breeding places, disinfestation of all air-craft, ships and railway trains, as also motor transport, should be maintained within flight range in our sea and air ports so that no imported mosquitoes can survive. Regulations framed on these lines and enforced properly, will prevent our malarial problem becoming still more complicated and will leave us free to tackle our existing ones.

Ordinances and Laws.—Legislative enactments are essential to give legal sanction for appropriate malaria-control measures within a country ; methods of irrigation, road construction, land cultivation and animal husbandry should be improved, so that they will tend to reduce rather than intensify the incidence of malaria. Enactments are also necessary to prevent malariogenic sites being utilised for housing projects. Bye-laws exist at present in all cantonment areas, and elaborate provision for anti-mosquito measures is contained in Municipal Acts, but the enforcement machinery everywhere is very slack. The entire scope and object of the bye-laws are thus ignored and we suffer continuous additions to our misery. The Model Mosquito Ordinance of the United States Public Health Service, the Straits Settlement Destruction of Mosquitoes Ordinance and the anti-mosquito provision in the Bombay Municipal Act have been published as appendices 15, 16 and 17 to the Bhore Committee's Report. All administrative bodies in India, (Central and States) should frame similar ordinances and bye-laws, modified where necessary to suit local conditions and *enforce them rigorously* within their respective jurisdictions.

Planning and organisation.—The WHO Expert Committee on Malaria has rightly stressed the fact that the effective control of malaria in any country is possible only by a properly established organised agency, say a Government controlled malaria organization of appropriate size, the personnel of which is suitably trained and adequately paid ; this requires the careful consideration of the Government of India especially in regard to the pay and prospects of the anti-malaria staff.

For success in a mosquito eradication project, preliminary information on several points is deemed essential by the WHO :— (*a*) the bionomics of the larvæ and adults of the vector species. (*b*) presence or absence of natural barriers, to prevent effectively the reintroduction of the eliminated species, as only then, the final objective of eradication can be attained ; (*c*) local topography ;

the breeding places of the vector or vectors should be readily accessible, since eradication depends mainly on anti-larval treatment; (*d*) local administrative conditions; for eradication has its own strategy requiring a detailed organisation with qualified personnel; (*e*) economic possibilities: for the cost of such a campaign may be the determining factor in the choice of the proper method. This means that any effective attack in a particular region must be based not only on what is known about the disease in general, but also upon its peculiar characteristics in that particular region.

A complete syllabus for the organisation of malaria-control on Central and State governmental lines by primary and secondary units has been framed by the Health Survey and Development Committtee set up in 1946; its recommendations though accepted in principle are still far from being adopted or enforced in practice. The selection and pay of staff for these organisations should follow the recommendations made by the Bhore Committee. Voluntary local help from the local inhabitants would be easy to obtain after sometime when the effects of Government control are clear. Filling of pits and the drainage campaign could be organised and carried out by labour, recruited from local volunteers and trained for a short time; this has already been tried and found successful in some areas. "Once the methods have been decided upon, eradication becomes an exercise in administration rather than an exclusively scientific or technical occupation," said Busvine.

It is possible that the Government, as has happened in some local bodies and even in some Central establishments, may decline to maintain a huge and expensive organisation for this purpose, after the disease has ceased to be a pressing public health problem; but such a step is false economy and might result in heavy losses at a future date. When communities begin to realise the seriousness of the disease and understand better the measures essential for the control of malaria, there is little doubt that both requisite funds and community support will be readily forthcoming. Upon these two factors therefore, depends the success or failure of antimalarial control measures.

Conclusion.—It has now been proved that malaria is quite vulnerable to planned attack and that the ultimate aim of any Government should be to eliminate this disease from being a public health menace to the country. As Haldane put it, "this will be a much more efficient measure of national defence than making ærodromes and warships". Malaria is slowly and steadily retreating dramatically as a result of the effective use of such powerful new insecticides as DDT; yet the disease is still very prevalent in large areas, causing misery and death to millions of its victims. Malaria makes food production impossibe in underdeveloped areas and seriously interferes with industrial and agricultural activities in

many tropical and sub-tropical areas; and it still takes a high toll of victims in infancy and early childhood". (WHO Expert Committee on Malaria).

"Each year brings fresh evidence to prove that mosquito-control is a sound investment, not only because of direct dividends, arising from elimination of the expense on illness and death, but also through bonuses earned by stimulated agriculture, trade, and property and by the general improvement of physical, social and intellectual welfare. Nothing which is subject to the will of the people costs a community more than preventable diseases and no expenditure returns to the people higher dividends than those invested in public health—a statement specially applicable to malaria." (Russel); and so malaria-eradication is an ideal worth achieving, although it may take a long time. The ideal is human and aims at saving humanity from misery, illness and death.

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### *Aspirin and Hæmatemesis*

Dr. Eric Frankel writes "During the last five months I have seen six patients under the age of 30 who were sent to hospital after a severe hæmatemesis, two requiring blood transfusions. None of these six people had any previous history of dyspeptic symptoms, and the hæmatemesis occurred spontaneously in the absence of evidence of other physical disease or mental disturbance. Every one of these six patients however, admitted to having *swallowed* one or more *whole tablets* of aspirin from half to three hours before they began to feel faint, developed nausea, and started to have a hæmatemesis. The aspirins were taken for muscular pain, headache and dysmenorrhœa. The patients made a rapid recovery on an ordinary gastric regime and had no further dyspeptic symptoms. Barium-meal examination two weeks after the hæmatemesis proved quite negative for gastric lesions The occurrence of gastric bleeding after the swallowing of aspirin must be a rare event, as aspirin is the most widely used analgesic drug which can be obtained by the general public without a medical prescription and *self-medication is the rule*. However, a few unfortunate susceptible people may suffer serious consequences if they *swallow the tablet whole on empty* stomach, and I should like to suggest that the manufacturers should label aspirin bottles and containers "*Crush before swallowing.*"—(*Br. Med. Jour.*, 4-8-1951, p. 302).

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### *Return of Vestibular Function following Streptomycin Toxæmia*

Of 62 patients with pulmonary tuberculosis, who were given 2 gm. of streptomycin daily for an average of 110 days, 42 had complete loss of vestibular response to caloric stimulation. In the great majority of cases response returned in varying degrees over a period of 2 years. More patients weighing less than 9 stones (57 kg.) lost response than did heavier patients and the rate of loss was also higher among patients aged over 45. Response returned earlier in the younger patients than in the older. Of 215 patients with pulmonary tuberculosis who were given only 1 gm. of streptomycin daily for an average of 110 days, 18 had a complete loss of vestibular response to caloric stimulation. Sixteen regained response within a year, one in 17 months and one was lost to study.—(*J.A.M.A.*, 146, 9, 1951, from *Calif. Med.*, 74, p. 185, 1951).

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(Spies & Bull in Duncan Diseases of Metabolism. 1947 p. 502)

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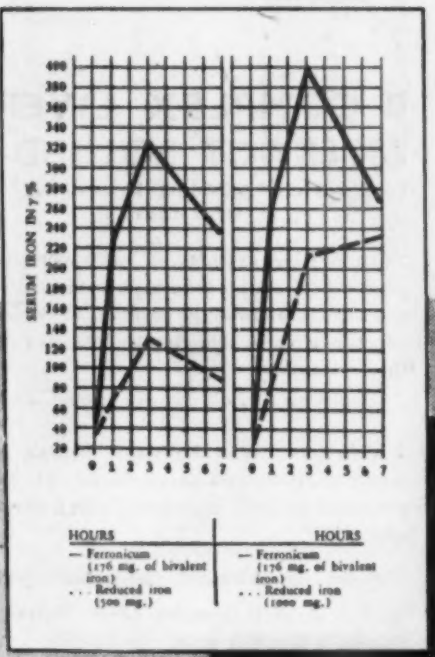
SERUM IRON IN γ %
HOURS
— Ferronicum (176 mg. of bivalent iron)
. . . Reduced iron (500 mg.)
HOURS
— Ferronicum (176 mg. of bivalent iron)
. . . Reduced iron (1000 mg.)

*The graph shows the better absorption of Ferronicum as compared to reduced iron. Small oral doses of Ferronicum increase the serum iron level far more than large doses of reduced iron (average from 5 patients). (Jasinsky, Schw. Med. Wschr., 80, 39, 1950)*

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# REPORT ON A STUDY OF THE VALUE OF ANTIBIOTICS IN CHOLERA

## (Terramycin, Chloromycetin and Aureomycin) and Formocibazol

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OUTBREAKS of epidemics, which were probably cholera, are described in Sanskrit literature as early as 400 B.C. Detailed accounts of this disease are however, available from about 1817, when a great epidemic occurred in this country. In the past there had been a great deal of confusion regarding the ætiology of cholera. As early as 1820, Jennison considered it to be due to a virus of an unknown nature, which caused purging and vomiting followed by shock due to a primary action on the stomach and intestine. He thought that cholera was not contagious and so quarantine was unnecessary in this disease. Reginal Orton attributed cholera to a deficient nervous action brought about by a lessened aeration of the blood which in turn depended upon unsettled weather conditions resulting from a deficiency of electrical fluid in the atmosphere! William Twining (1833) also an advocate of the nerve-theory, laid stress on the relation of symptoms to cessation of functions of the organs supplied by the great solar-plexus! He made an admirable clinical study of this disease and described clearly the treatment in its different stages; he also refuted the theory of venesection which was strongly advocated by certain physicians of the time, *e.g.*, Frank Corbyn (1832). Corbyn's work is noteworthy, for he strongly advocates the use of large doses of calomel and opium in combination with venesection, while he severely condemned the use of stimulants. He agreed with Jennison, Orton and Twining, that climatic variations and chills played an important role in cholera, but he attributed its capricious distribution to local generation of the poison due to a peculiar state of the soil as suggested by Sydenham. Corbyn thought that the poison acted by suddenly checking perspiration thus inducing internal congestion followed by inflammation. Thus, the ætiology of cholera remained a mystery until 1833, when during the 5th pandemic, Robert Koch described for the first time the comma-shaped bacillus as the causative organism of cholera.

More than a century has elapsed since Koch discovered the cause, but very little advance has been made so far as the treatment of cholera is concerned. The sulphonamides opened up a new field for research in this disease. They were tried by Chopra *et al*[4] (1941), Carruthers[2] (1942), Griffiths[7] (1942), Sadusk and Oswald[10] (1943), Bhatnagar *et al*[7] (1948) and Lahiri[8] (1945, 1948, 1951). The discovery of the antibiotics, chloromycetin, aureomycin and terramycin have brought about a revolutionary change in the conceptions regarding the treatment of many diseases. Gauld *et al*[7] (1949) by means of experiments both *in vivo* and *in vitro*, showed that chloromycetin completely inhibited the growth of vibrio cholerae. Gauld's observation prompted Chaudhuri *et al*[3] (1950) to undertake a clinical trial but finding no difference in the course of the disease they suggested further investigation. Terramycin was tried in cholera by Das *et al*[5] (1951), and Konar and Sengupta[8] (1951) suggested the use of this drug for reducing the number of carriers.

The physician today is quite at a loss to understand whether chemotherapeutic agents and antibiotics really play an important role in the treatment of cholera. In this paper, we have undertaken a detailed study regarding the action of antibiotics (terramycin, chloromycetin, aureomycin) and formocibazol in cholera. One hundred and sixty cases have been studied here, out of which one hundred and seven were males and fifty three females, their ages varying between 2 and 68 years. We would like to mention here that the antibiotics were used in these cases as an adjuvant to the routine treatments like saline and circulatory stimulants, etc.

Experiments and observations.—The trials of these drugs were undertaken in the Cholera Wards of the R. G. Kar Medical College and Hospitals from March to June, 1952. After admission, the degree of dehydration was noted, slight being expressed as +, moderate as ++, severe as +++. Sterile bed-pans were kept ready and patients were allowed to evacuate in these. Material from fæces was directly inoculated at the bedside into alkaline fluid media and were then transferred to other solid media such as taurocholate agar, S.S. agar and desoxycholate citrate agar, in order to minimise false negatives as far as possible. Of the strains isolated two belonged to the Inaba and the rest to the Ogawa types.

The stool cultures were done every day until the patients remained in the hospital. In cases where evacuation stopped and no fæcal matter could be obtained, rectal swabs were taken and media inoculated. For comparison the stool cultures of 50 patients (cases 1 to 50) to whom no antibiotic was administered were also examined every day until they left the hospital, and they served as control. Terramycin was tried in 40 cases (Nos. 51 to 90), chloromycetin in 20 cases (Nos. 91 to 110), aureomycin in 20 cases (Nos. 111 to 130) and formocibazol in 30 cases (Nos. 131 to 160). The dosage was very small (as compared to that used by other workers) in the case

of terramycin, chloromycetin and aureomycin; it was 250 mg. every 6 hours, while in the case of formocibazol 4 tablets were given first and then 2 tablets every 4 hours. Very little water was allowed with the drugs in order to minimise vomiting. But if, inspite of this precaution, vomiting occurred the dose was repeated. The drugs were administered for at least 48 or 72 hours and in some cases for a longer time according to the condition of the patient. For convenience, cases whose stool cultures were negative have not been recorded here and we have also not taken those cases into consideration for the assay, which proved fatal within the first 24 hours of admission, because in these cases very little antibiotic could be administered before death.

We observed that in the control group (Nos. 1 to 50) stool cultures became negative usually between the 8th and 15th day though a few variations were noted. In the terramycin group (Nos. 51 to 90) stool cultures were negative as a rule on the 2nd day *i.e.*, within 24 hours of starting the antibiotic. In the chloromycetin group (Nos. 91 to 110) and aureomycin group (Nos. 111 to 130) the stools became negative usually between the 2nd and 5th days. In cases (Nos. 130 to 160) where formocibazol was administered the stools were usually negative between three and six days. For ease of reference, the cases are tabulated in the accompanying tables. The deaths in the control group of 50 cases were 22%, in the terramycin group of 40 cases 10%, in the aureomycin group of 20 cases 10%, in the chloromycetin group of 20 cases 15%, and in the formocibazol group of 30 cases 20%. As regards other points *e.g.*, appearance of bile in the stool and the re-appearance of solid stool, practicallly no difference was noted in the different groups.

**Experiments in vitro**

| Antibiotic | Size of zone (average) | Strength of antibiotic solution |
|---|---|---|
| Terramycin | 19 m.m. | 1 microgram per c.c. |
| Chloromycetin | 19 m.m. | 1 microgram per c.c. |
| Aureomycin | 20 m.m. | 1 microgram per c.c. |
| Formocibazol | 19 m.m. | 5 mg. per cent. |

The above statement shows the average size of the inhibitory zones with the antibiotics and with formocibazol.

In the *in vitro* tests all the strains were found equally susceptible to formocibazol, terramycin, aureomycin and chloramphenicol. They were tested by the Wilkin's cup method and the size of the zones measured in each case. A mercuric chloride control was set up with each plate for better comparison.

| Serial No. | Name | Age | Sex | Pulse-rate | Degree of dehydration | Group | Stool culture before administration of antibiotic. | Day on which stools became negative | Appearance of bile in the stools (in days). | Appearance of solid stools (in days) | Result | Complication |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Yrs. | | | | | | | | | | |
| 1 | L | 20 | F | Imp | + + + | Control | V. Comma. | 8th day | 1 | 5 | Cured | |
| 2 | A | 35 | M | 96 | ,, ,, | do | do | 9th day | 2 | 6 | ,, | |
| 3 | N | 26 | M | 104 | ,, ,, | do | do | 7th day | 2 | ... | ,, | |
| 4 | R | 14 | M | 112 | ,, ,, | do | do | ... | 2 | ... | Died of uræmia on 4th day | |
| 5 | P | 8 | M | 98 | ,, ,, | do | do | 7th day | 1 | 4 | Cured | |
| 6 | N | 34 | M | 126 | ,, | do | do | 8th day | 1 | 5 | ,, | |
| 7 | A | 38 | M | imp | ,, ,, ,, | do | do | 13th day | 1 | ... | ,, | |
| 8 | B | 40 | M | ,, | ,, ,, | do | do | 8th day | 2 | 7 | ,, | broncho-pneumonia |
| 9 | K | 30 | F | ,, | ,, ,, ,, | do | do | 10th day | 2 | 6 | ,, | |
| 10 | M | 30 | M | ,, | ,, ,, ,, | do | do | ... | 1 | ... | Died of uræmia on 5th day | |
| 11 | S | 10 | M | ,, | ,, | do | do | 15th day | 2 | 4 | Cured | |
| 12 | R | 2 | M | ,, | ,, ,, ,, | do | do | 9th day | 1 | 7 | ,, | |
| 13 | K | 38 | M | 100 | ,, ,, | do | do | 8th day | 2 | 4 | ,, | |
| 14 | A | 24 | F | imp | ,, ,, ,, | do | do | 10th day | 1 | 5 | ,, | herpes labialis |
| 15 | K | 18 | M | 120 | ,, ,, | do | do | 14th day | 1 | 3 | ,, | |
| 16 | H | 6 | M | imp | ,, ,, ,, | do | do | ... | ... | ,, | Died of peripheral circulatory failure on 2nd day | |
| 17 | N | 50 | M | 82 | ,, | do | do | 8th day | 1 | 4 | Cured | |
| 18 | B | 52 | M | imp | ,, ,, ,, | do | do | 15th day | 2 | 5 | ,, | uræmia |
| 19 | D | 23 | M | ,, | ,, ,, | do | do | 7th day | 2 | 6 | ,, | |
| 20 | T | 7 | M | 110 | ,, | do | do | 8th day | 2 | ,, | ,, | herpes labialis |
| 21 | P | 46 | M | imp | ,, ,, ,, | do | do | 15th day | 1 | 4 | ,, | |
| 22 | N | 21 | F | ,, | ,, ,, ,, | do | do | 7th day | 1 | 5 | ,, | |
| 23 | T | 32 | F | ,, | ,, ,, | do | do | ... | 2 | ... | Died of uræmia on 5th day | |
| 24 | P | 28 | F | ,, | ,, ,, ,, | do | do | ... | ... | ... | Died of peripheral circulatory failure on 2nd day | |
| 25 | A | 9 | F | 126 | ,, ,, | do | do | 9th day | 1 | 7 | Cured hyperpyrexia. | |
| 26 | K | 47 | M | imp | ,, ,, | do | do | 9th day | 1 | 8 | ,, | |
| 27 | S | 39 | F | 88 | ,, | do | do | ... | 2 | ... | Died of uræmia on 4th day | |
| 28 | T | 24 | M | 130 | ,, | do | do | 15th day | 2 | 4 | Cured | |
| 29 | P | 17 | M | imp | ,, | do | do | 15th day | 2 | 6 | ,, | |
| 30 | K | 22 | F | ,, | ,, ,, ,, | do | do | 13th day | 1 | 5 | ,, | |
| 31 | R | 27 | M | 102 | ,, | do | do | 13th day | 1 | 6 | ,, | |
| 32 | A | 49 | M | imp | ,, ,, ,, | do | do | 15th day | 1 | 7 | ,, | |
| 33 | A | 40 | M | ,, | ,, ,, ,, | do | do | 11th day | 2 | ... | ,, | |
| 34 | N | 43 | F | 98 | ,, | do | do | 7th day | 3 | 8 | ,, | |
| 35 | A | 14 | F | 116 | ,, | do | do | 9th day | 2 | 4 | ,, | |
| 36 | P | 25 | M | 127 | ,, ,, | do | do | ... | .. | ... | Died of uræmia on 3rd day | |
| 37 | S | 12 | M | imp | ,, ,, ,, | do | do | 8th day | 2 | 5 | Cured | |
| 38 | K | 39 | M | ,, | ,, ,, ,, | do | do | 12th day | 2 | 5 | ,, | |
| 39 | R | 21 | M | ,, | ,, ,, ,, | do | do | ... | ... | ,, | Died of peripheral circulatory failure on 2nd day | |
| 40 | R | 22 | F | 112 | ,, | do | do | 6th day | 3 | 7 | Cured | |
| 41 | D | 53 | M | imp | ,, ,, ,, | do | do | 7th day | 2 | ... | ,, | |
| 42 | P | 44 | M | ,, | ,, ,, | do | do | 6th day | 2 | 4 | ,, | Uræmia |
| 43 | A | 44 | M | ,, | ,, ,, | do | do | 13th day | 2 | 5 | ,, | |

| Serial No. | Name | Age | Sex | Pulse rate | Degree of dehydration | Group | Stool culture before administration of antibiotic | Day on which stools become negative | Appearance of bile in the stools (in days) | Appearance of solid stools (in days) | Result | Complication |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 44 | S | 30 | F | imp | +++ | Control | V. Comma. | 15th day | 1 | .. | Cured | |
| 45 | B | 24 | M | 108 | ,, | do | do | 14th day | 2 | 6 | ,, | |
| 46 | G | 18 | M | imp | ,, . | do | do | 15th day | 2 | 7 | ,, | |
| 47 | F | 22 | F | ,, | ,, ,, ,, | do | do | .. | 1 | .. | Died of peripheral circulatory failure on 2nd day | |
| 48 | M | 11 | M | ,, | .. ,, | do | do | .. | 3 | .. | Died of uræmia on 4th day | |
| 49 | P | 41 | M | 116 | ,, ,, | do | do | 10th day | 2 | 4 | Cured | |
| 50 | K | 27 | M | imp | ,, ,, ,, | do | do | ... | .. | .. | Died of peripheral circulatory failure on 2nd day | |
| 51 | G | 27 | F | ,, | ,, ,, | Terramycin | do | 2nd day | 3 | 4 | Cured | |
| 52 | P | 22 | F | ,, | ,, ,, ,, | do | do | do | 2 | 4 | ,, | |
| 53 | A | 58 | M | ,, | ,, ,, | do | do | do | 2 | 6 | ,, Hyperpyrexia | |
| 54 | S | 33 | M | ,, | ,, ,, ,, | do | do | do | 1 | 4 | ,, | |
| 55 | A | 30 | F | ,, | ,, | do | do | do | 1 | 4 | ,, | |
| 56 | B | 21 | M | ,, | ,, ,, | do | do | do | 2 | 5 | ,, | |
| 57 | G | 53 | F | 114 | ,, | do | do | do | 2 | 3 | ,, | |
| 58 | S | 42 | M | 116 | ,, ,, | do | do | do | 2 | 3 | ,, | |
| 59 | H | 63 | F | imp | ,, ,, ,, | do | do | do | 1 | .. | Died of uræmia on 4th day | |
| 60 | M | 6 | F | ,, | ,, ,, | do | do | do | 1 | 4 | Cured | |
| 61 | R | 28 | M | ,, | ,, ,, ,, | do | do | do | 1 | 5 | ,, Herpes labialis | |
| 62 | S | 11 | M | 106 | ,, | do | do | do | 1 | 7 | ,, | |
| 63 | M | 40 | M | imp | ,, ,, ,, | do | do | do | 1 | 5 | ,, | |
| 64 | P | 14 | M | ,, | ,, ,, | do | do | do | 1 | 4 | ,, | |
| 65 | M | 12 | M | 134 | ,, ,, | do | do | do | 2 | 7 | ,, | |
| 66 | D | 19 | F | imp | ,, ,, ,, | do | do | do | 1 | 5 | ,, | |
| 67 | D | 21 | M | ,, | ,, ,, ,, | do | do | do | 2 | .. | Died of uræmia on 3rd day | |
| 68 | N | 38 | F | 98 | ,, | do | do | do | 1 | 6 | Cured | |
| 69 | P | 40 | M | imp | ,, ,, ,, | do | do | do | 1 | 5 | ,, | |
| 70 | R | 40 | M | ,, | .. ,, | do | do | do | 2 | 4 | ,, | |
| 71 | P | 26 | F | ,, | ,, ,, | do | do | do | 1 | 3 | ,, | |
| 72 | J | 30 | M | 116 | ,, ,, | do | do | do | 1 | .. | ,, | |
| 73 | M | 45 | M | imp | ,, ,, ,, | do | do | do | 2 | 4 | ,, | |
| 74 | R | 18 | M | ,, | ,, ,, ,, | do | do | do | 2 | 5 | ,, | |
| 75 | P | 6 | M | ,, | ,, ,, ,, | do | do | do | 3 | 7 | ,, Herpes labialis | |
| 76 | D | 16 | M | 122 | ,, ,, | do | do | do | 1 | 6 | ,, | |
| 77 | M | 22 | F | imp | ,, ,, | do | do | do | 1 | 9 | ,, | |
| 78 | R | 23 | M | ,, | ,, ,, ,, | do | do | do | 1 | 4 | ,, | |
| 79 | P | 14 | F | ,, | ,, ,, | do | do | do | 2 | 4 | ,, | |
| 80 | A | 62 | F | ,, | ,, ,, ,, | do | do | do | 2 | 6 | ,, | |
| 81 | J | 38 | M | ,, | ,, ,, | do | do | do | 3 | 6 | ,, | |
| 82 | P | 37 | M | ,, | ,, ,, ,, | do | do | do | 2 | 5 | ,, | |
| 83 | B | 35 | M | ,, | ,, ,, ,, | do | do | do | 1 | .. | Died of uræmia on 3rd day | |
| 84 | N | 26 | F | ,, | ,, ,, | do | do | do | 2 | 4 | Cured | |
| 85 | S | 10 | M | ,, | ,, ,, ,, | do | do | do | 1 | 5 | ,, | |
| 86 | A | 29 | M | ,, | ,, ,, | do | do | do | 2 | 4 | ,, | |
| 87 | M | 44 | F | ,, | ,, ,, ,, | do | do | do | 2 | .. | Died of peripheral circulatory failure on 2nd day | |

| Serial No. | Name | Age | Sex | Pulse rate | Degree of dehydration | Group | Stool culture before administration of antibiotic | Day on which stools became negative | Appearance of bile in the stools (in days) | Appearance of solid stools (in days) | Result | Complication |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 88 | R | 23 | M | 134 | + + | Terramycin | V. Comma | 2nd day | 2 | 4 | Cured | |
| 89 | J | 45 | F | imp | + + + | do | do | do | 2 | 4 | ,, | |
| 90 | R | 32 | M | ,, | ,, ,, ,, | do | do | do | 2 | 5 | ,, | |
| 91 | M | 21 | M | 126 | ,, ,, | Chloromycetin | do | do | 3 | ... | ,, | |
| 92 | H | 54 | M | imp | ,, ,, ,, | do | do | 3rd day | 2 | 4 | ,, | |
| 93 | S | 44 | F | ,, | ,, ,, | do | do | do | 1 | 5 | ,, | |
| 94 | A | 59 | M | ,, | ,, ,, ,, | do | do | do | 1 | .. | Died of uræmia on 3rd day | |
| 95 | S | 12 | M | ,, | ,, ,, | do | do | 4th day | 2 | 4 | Cured | |
| 96 | K | 4 | M | ,, | ,, ,, ,, | do | do | ... | 2 | ... | Died of peripheral circulatory failure on 2nd day | |
| 97 | C | 13 | M | ,, | ,, ,, ,, | do | do | 3rd day | 2 | 8 | Cured | |
| 98 | N | 47 | M | ,, | ,, ,, | do | do | 4th day | 2 | 6 | ,, | |
| 99 | S | 38 | M | 108 | ,, | do | do | 3rd day | 3 | 4 | ,, | |
| 100 | B | 16 | M | imp | ,, ,, ,, | do | do | 2nd day | 2 | 5 | ,, | |
| 101 | P | 21 | M | 130 | ,, ,, | do | do | do | 2 | 5 | ,, | |
| 102 | T | 8 | M | imp | ,, ,, | do | do | .. | 2 | .. | Died of peripheral circulatory failure on 3rd day | |
| 103 | K | 11 | F | ,, | ,, ,, ,, | do | do | 3rd day | 2 | 4 | Cured | |
| 104 | P | 17 | F | ,, | ,, ,, ,, | do | do | do | 1 | 6 | ,, | |
| 105 | F | 65 | M | 102 | ,, | do | do | do | 1 | 6 | ,, | |
| 106 | A | 68 | F | imp | ,, ,, | do | do | 2nd day | 2 | 4 | ,, | |
| 107 | M | 45 | F | 94 | ,, | do | do | do | 1 | 4 | ,, | |
| 108 | P | 60 | M | imp | ,, ,, ,, | do | do | 3rd day | 2 | 5 | ,, | |
| 109 | A | 48 | F | ,, | ,, ,, ,, | do | do | do | 2 | 5 | ,, | |
| 110 | S | 40 | M | ,, | ,, ,, | do | do | 4th day | 1 | 7 | ,, | |
| 111 | B | 40 | M | ,, | ,, ,, ,, | Aureomycin | do | do | 1 | 5 | ,, | |
| 112 | C | 21 | M | 134 | ,, ,, | do | do | 3rd day | 2 | 6 | ,, | |
| 113 | H | 20 | M | imp | ,, ,, ,, | do | do | do | 2 | 7 | ,, | |
| 114 | K | 40 | F | ,, | ,, ,, ,, | do | do | 2nd day | 2 | 6 | ,, | |
| 115 | H | 40 | M | ,, | ,, ,, | do | do | 4th day | 2 | 6 | ,, Uræmia | |
| 116 | A | 30 | M | ,, | ,, ,, ,, | do | do | 3rd day | 1 | 8 | ,, | |
| 117 | A | 20 | F | 110 | ,, | do | do | do | 1 | .. | ,, | |
| 118 | S | 60 | M | 120 | ,, ,, | do | do | do | 1 | 4 | ,, | |
| 119 | K | 10 | F | imp | ,, | do | do | ... | ... | ... | Died of hyperpyrexia on 2nd day | |
| 120 | K | 20 | F | ,, | ,, ,, ,, | do | do | 4th day | 1 | ... | Cured | |
| 121 | U | 40 | M | ,, | ,, ,, | do | do | 3rd day | 2 | 5 | ,, | |
| 122 | K | 18 | M | ,, | ,, ,, | do | do | do | 2 | 7 | ,, | |
| 123 | R | 40 | M | 130 | ,, | do | do | do | 2 | 6 | ,, | |
| 124 | A | 40 | M | imp | ,, ,, ,, | do | do | 4th day | 1 | 5 | ,, | |
| 125 | R | 35 | M | ,, | ,, ,, | do | do | ... | 2 | ... | Died of peripheral circulatory failure on 3rd day | |
| 126 | F | 20 | M | ,, | ,, ,, ,, | do | do | 3rd day | 1 | 6 | Cured | |
| 127 | J | 52 | F | ,, | ,, ,, ,, | do | do | 4th day | 1 | 4 | ,, | |
| 128 | S | 28 | M | ,, | ,, ,, ,, | do | do | 3rd day | 2 | 7 | ,, | |
| 129 | S | 34 | M | ,, | ,, ,, | do | do | do | 1 | .. | ,, | |
| 130 | K | 40 | F | 122 | ,, ,, | do | do | do | 1 | 6 | ,, | |
| 131 | J | 30 | F | imp | ,, ,, ,, | Formocibazol | do | do | 1 | 7 | ,, | |
| 132 | F | 35 | M | ,, | ,, ,, ,, | do | do | ... | .. | .. | Died of hyperpyrexia on 2nd day | |

| Serial No. | Name | Age | Sex | Pulse rate | Degree of dehydration | Group | Stool culture before administration of antibiotic. | Day on which stools became negative | Appearance of bile in the stools (in days) | Appearance of solid stools (in days) | Result | Complication |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 133 | S | 61 | M | 98 | + | Formocibazol | V. Comma | 5th day | 2 | 4 | Cured | |
| 134 | B | 23 | M | imp | + + + | do | do | 4th day | 1 | 5 | ,, | |
| 135 | H | 14 | F | ,, | ,, ,, | do | do | 5th day | 1 | 6 | ,, | |
| 136 | K | 20 | M | ,, | ,, ,, ,, | do | do | 5th day | 2 | 7 | ,, | |
| 137 | S | 20 | M | ,, | ,, ,, | do | do | 4th day | 1 | ... | ,, | |
| 138 | M | 30 | M | 134 | ,, ,, ,, | do | do | 4th day | 1 | 6 | ,, | |
| 139 | P | 40 | F | imp | ,, ,, | do | do | 3rd day | 3 | 8 | ,, | |
| 140 | M | 42 | M | ,, | ,, ,, ,, | do | do | 4th day | 2 | 9 | ,, | |
| 141 | M | 56 | M | ,, | ,, ,, ,, | do | do | ... | 2 | ... | Died of uræmia on 7th day | |
| 142 | R | 9 | M | ,, | ,, ,, ,, | do | do | ... | 3 | ,, | Died of peripheral circulatory failure on 4th day | |
| 143 | A | 17 | F | 118 | ,, ,, | do | do | 6th day | 1 | 5 | Cured | |
| 144 | A | 18 | M | imp | ,, ,, ,, | do | do | 4th day | 1 | 7 | ,, | |
| 145 | E | 48 | F | ,, | ,, ,, ,, | do | do | 5th day | 2 | 8 | ,, Uræmia | |
| 146 | F | 40 | F | ,, | ,, ,, | do | do | 5th day | 2 | 6 | ,, | |
| 147 | R | 52 | M | ,, | ,, ,, | do | do | 6th day | 2 | ,, | ,, | |
| 148 | R | 40 | M | ,, | ,, ,, ,, | do | do | 5th day | 2 | 4 | ,, | |
| 149 | D | 20 | M | ,, | ,, ,, ,, | do | do | 4th day | 2 | ,, | ,, | |
| 150 | B | 24 | M | ,, | ,, ,, ,, | do | do | ... | 1 | ,, | Died of peripheral circulatory failure on 3rd day | |
| 151 | R | 30 | F | ,, | ,, ,, ,, | do | do | 5th day | 1 | 9 | Cured | |
| 152 | P | 30 | F | ,, | ,, ,, ,, | do | do | 6th day | 1 | 7 | ,, | |
| 153 | M | 11 | M | 112 | ,, ,, | do | do | 6th day | 2 | ,, | ,, | |
| 154 | T | 29 | M | imp | ,, ,, ,, | do | do | ... | ... | ... | Died of hyperpyrexia on 3rd day | |
| 155 | J | 34 | M | ,, | ,, ,, ,, | do | do | 4th day | 1 | 6 | Cured | |
| 156 | R | 16 | M | ,, | ,, ,, | do | do | 5th day | 1 | 5 | ,, | |
| 157 | K | 55 | M | ,, | ,, ,, ,, | do | do | 3rd day | 1 | 6 | ,, | |
| 158 | K | 30 | F | ,, | ,, ,, | do | do | 5th day | 1 | 7 | ,, | |
| 159 | T | 40 | M | ,, | ,, ,, ,, | do | do | ... | 2 | ... | Died of uræmia on 5th day | |
| 160 | M | 30 | F | ,, | ,, ,, ,, | do | do | 5th day | 1 | 5 | Cured | |

Conclusion.—In *in vitro* experiments, formocibazol, terramycin, chloromycetin, and aureomycin showed very little difference in their antibacterial activity against both Inaba and Ogawa strains of the vibrio cholerae.

The cultural results showed that in spite of the smaller dosage used by us (250 mg. every 6 hours in the case of antibiotics and 4 tablets *stat* and 2 tablets every four hours in the case of formocibazol) as compared to that of other workers, terramycin invariably rendered the stools negative earlier *i.e.*, within 24 hours as compared to other antibiotics.

The mortality in the 40 patients treated with terramycin was 10%; in the 20 patients of the chloromycetin group it was 15%, in the 20 patients of the aureomycin group it was 10%, and in 30 patients of the formocibazol group it was 20%, as compared with

the control group which showed a mortality of 22 %. This is however, of no statistical value whatsoever; and it was not at all possible to assess the relative merits of these drugs as far as their curative value in cholera was concerned because the number was not sufficiently large and of the same severity in all cases and so not strictly comparable. But it was noted that with the antibiotics, the mortality was markedly less as compared with the control group and so it cannot be said that the antibiotics had absolutely no curative value as judged by the results of the present study.

A reduction in the number of carriers through a sterilization of the gut by the antibiotics, naturally brings us to the question of whether these drugs could be used in the prophylaxis of cholera among this contacts and among the general population exposed to infection during an epidemic, in addition to or as an alternative to the cholera inoculations. This is obviously important from the public health point of view and we would request the health authorities to investigate the prophylactic value if any, of these antibiotics in controlling epidemics of cholera.

**Acknowledgement.**—We are indebted to the authorities of the R. G. Kar Medical College Hospitals for the facilities kindly given to us for carrying out this work. Our thanks are due to Dr. Ajit Roy and Dr. Amal Dutta for the collection of the material and to Messrs. Parke Davis & Co., Ciba Pharma Ltd., and Dey's Medical Stores Ltd. (Exclusive distributors of Chas Pfizer Co.) for a free supply of the drugs used in this investigation.

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### *Terramycin in Treatment of Pneumonias of Childhood*

Wooding and Scott of the Department of Pædiatrics of the Howard University School of Medicine, Washington, report on 10 cases of bacterial pneumonia due to pneumococci, staphylo or streptococci, which were treated with terramycin. Two of these had pertussis also. The ages of the patients varied from 4 months to 8 years. The dose of terramycin was 1·8 to 4·5 gm. daily and the treatment was on an average for 6 days. Nine cases responded promptly, the temperature falling by immediate crisis. Terramycin is certainly of great value in pneumonias of childhood, as its action covers a wide anti-bacterial spectrum and it is effective when given orally. It is also considered a distinct advantage to have at hand a substance available for treating patients who may be sensitive to other antibiotics in common use.—(*Jour. Paediat.*, 38, 423-27, 1951).

# HEPATO-SPLENOMEGALY OF UNDETERMINED ETIOLOGY—LIPOIDOSES?

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THE following case-histories of two brothers are worthy of careful study.

1. K.M. 2. S.M.
Lipoidoses?

1. K. M., 11 years old. Male (Mohamadan). Admitted into the Government Stanley Hospital, Madras, on 9-9-1941 and discharged on 13-10-1941.

Complaint.—Fever and distension of abdomen for one year.

*Previous history*:—Mother's pregnancy and labour normal. Had fever and fits soon after birth; vaccinated before end of first year. Accustomed to a mixed diet.

*Family history*:—Parents are first cousins. Family of 6 children: (1) male 14 years old. Liver and spleen not enlarged. (2) The patient. (3) Male 8 years old. Liver 1" below costal margin. (4) Male 6 years old. Liver and spleen not enlarged. (5) Female 5 years old. Liver and spleen not enlarged. (6) Male. Died of fever at 3 months?

*History of present illness*:—Used to get attacks of fever now and then for the past one year. Abdomen gets bloated; bowels constipated; micturition normal. Treated for kala azar prior to admission into hospital.

CLINICAL NOTES:—A boy with hyperteloric facies; not anæmic; lumbo-dorsal kyphosis present; conjunctival xerosis present; height 3' 11¼"; weight 42 lbs; speech thick; gait normal; no osseous abnormalities. Mentally backward (?) afraid of drums and aeroplanes. Lymph glands large in the groin and axillae.

Abdomen: Protuberant. Liver $3^1/_2$" below costal margin, and ishard. Spleen 5" below costal margin and hard. Circumference of the abdomen around umbilicus 23". No free fluid present inside abdomen. Heart and lungs: nil particular.

Eyes: Fundus examination showed chloestrin [?] crystal deposits particularly in the outer and upper quadrants.

*Investigations*:—Urine: Sp. Gr. 1010. Acid in reaction. Albumin and sugar absent. No pus cells. Culture for B. coli:—negative.

Blood:—No malarial parasite. Widal: negative 1 in 25. Kahn and Wassermann: both negative. Takata Ara: moderately positive. Gel and Chopra test: negative. Sedimentation rate: 45 mm. in the hour. liver puncture: negative for Leishman-Donovan bodies. Red cells: 3·7 millions. Hæmoglobin: 70%. Blood platelets: 170000 per cmm. Reticulocytes: 1%. Whites: 3000. Colour index: 0·95. Differentially whites:—P. 42·5%, L. 51%, M. 6%, E. 0·5; Mast 0%. No immatures. 9-10-'41 Blood cholesterol 98 mg. % and on 23-9-'41 108 mg. %. Blood culture: no growth. Van den Bergh: direct and indirect both negative. Fragility begins at 0·4% and definite at 0·34%.

X-rays: spine and skull appear normal.

Circumference of the head touching the supraorbital ridges and external occipital protuberance is 19¼". Distance between ears over vertex is 11¼".

COMMENT.—The presence of hyperteloric facies and mental deficiency, protuberant abdomen with hepato-splenomegaly, lead to a consideration of storage deficiency diseases like Gauchers disease, Niemann-Pick disease, Schuller-Christian syndrome, Von-Gierke's disease and also Gargoylism. Enlarged liver and spleen rule out Morqio's disease, and splenomegaly rules out again Von Gierke's disease. Malaria, congenital syphilis, Kala azar and Banti's syndrome have been ruled out by the investigations. Fundus infiltration with cholesterol crystals suggests lipoidosis.

2. S.M., male, 8 years. (Brother of K.M.) Admitted on 23-9-'41 and discharged on 9-10-'41. Discovered on a routine examination of all the children in the family.

A boy of average nutrition. Intelligence appears unimparied. Teeth and tongue clean. Hyperteloric facies present. Circumference of the head touching the external occipital protuberance and supraorbital margins is 19¼". Distance between ears over vertex is 11¾". Abdomen: Liver 1½" below costal margin. Spleen ½". Heart and Lungs: nil particular. X-rays wrist: 3 centres of ossification present, while the age is 8 years. X-rays spine:—no deformities seen. Fundus:—cholestrin deposits present in both eyes.

Thus the patient is like his brother in the manifestations.

COMMENT.—The brothers described above are probably examples of an undetermined type of *Lipoidosis*.

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## CHILD WELFARE

### PÆDIATRICIANS IN CONFERENCE

INAUGURATING the third "All-India Pædiatric Conference at the Madras Medical College, on Sunday the 24th August '52, the Governor of Madras Shri SRI PRAKASA called on medical men to pay serious attention to the "alarming rise in the population", and to the proper training of the children in order to make them grow into healthy and useful citizens of the sovereign independent Republic. Dr. M. B. PRABHU who presided over the conference also pleaded for fostering in children ideas of co-operation and fellow feeling by instilling in them better values of life. Dr. A. L. MUDALIAR the Vice-Chancellor of the Madras University welcomed the delegates who had come from all over India and from Ceylon and said that child welfare clinics, child guidance clinics, schools for the defective and physically disabled children, homes for the problem-children and orphanages should all be run in an integrated manner along with the nursery schools and school medical inspection programmes. He referred to the dangers of the so-called specialisation in medicine and to the movement now fortunately spreading in the western countries towards developing the general practitioner and the family doctor to come into their own and take up the treatment of all conditions including pædiatrics, atleast in the first instance. He warned the pædiatricians against taking a narrow or myopic view of the wider issues involved in child care and child welfare. They should remember that their speciality needed a good grasp of child psychology, and of the methods to be adopted for a simultaneous improvement of the physical and mental conditions of the child. They were specialists only as regards a particular age-period in the life of their patients and

not as regards a particular anatomical structure or a specific physiological function. Speaking from personal experience of many years, as one intimately connected with and concerned about the training given to the medical students and doctors in general practice or in specialised studies, he considered that pædiatrics had received only a step-motherly treatment from educationists in many countries. "There could be no better initiation into the study of medicine than through the door of pædiatrics and it was his hope" he said, "that in the revised regulations that had been drawn up by the Indian Medical Council and which the Madras University would also be adopting, the under-graduate would have greater opportunities to study medicine and social medicine through children's clinics and children's wards". He pleaded next for a compulsory post-graduate training in pædiatrics for atleast six months for those who wished to take the higher degree of Doctor of Medicine. This is indeed a most desirable and welcome procedure and there can be no objection to its inclusion in the curriculum of studies for the M.D. Degree. Dr. PRABHU in his presidential address suggested that refresher courses should be organized for the general practitioners and that the rules framed therefor should be sufficiently comprehensive and elastic so as to afford the G.Ps. every encouragement and facility. Speaking about rural welfare, he said that in any scheme of things for the care of our children the rural areas should not be ignored or over-looked. The largest number, nearly 80 percent and more, resided in rural areas and the economic, social, sanitary and literary standards were very unsatisfactory there. A beginning had to be made to open in each village, a *creche* where the children could be collected and looked after, while the parents went out for work. The mobile Health Units should visit these villages and afford all help. The villager should not have to leave his village and go a long way to obtain his bare requirements; on the other hand, his medical requirements should be taken to him. Dr. CHAUDHURI, a leading pædiatrician of Bengal and ex-President of the Society expressed the hope that every one will co-operate in this great task of caring for the children. Shri SHETTY, the Madras Minister of Health said that the problem of rural medical relief was a head-ache to his government but they were trying their best to devise means for affording relief to the rural population; he pleaded for more women doctors taking to specialised work, particularly pædiatrics, than men doctors.

Nearly all the speakers at this important Conference, felt that there had been in recent years, undue emphasis on specialisation, which was detrimental to the interests of the patients, as well as to the general usefulness of the family doctor. "The knowledge of the specialist about the whole human system," Dr. MUDALIAR humorously remarked, "was sometimes in inverse proportion to the sphere of his extreme specialisation." We certainly do not mean

to say that there should be no specialisation, but we certainly plead for every one of the general practitioners acquiring knowledge, if necessary by a short course of training in the specialities, so that his roles as a general practitioner, able to deal with and treat abnormal conditions and as a family doctor may be re-established. He will diagnose and treat not only the disease but also the patient whose antecedents and family history are sufficiently well known to him ; and will take consultations with a specialist, only when he considers it imperative in the interests of the patient or when the patient insists that he should be seen by a specialist. Patients requiring constant specialist attention and treatment would of course, be passed on by him to the respective specialists or institutions, but he should still keep a watch over the patient's treatment, progress and convalescence. This procedure is particularly necessary in the case of children and their ailments. So we heartily approve of the resolutions and recommendations adopted at this Conference of Pædiatricians.

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## MINISTERIAL PRONOUNCEMENTS OF MOMENT

### (CONFERENCE OF DISTRICT MEDICAL OFFICERS)

THE month of August 1952 in Madras has been a very eventful one ; the attention of the top-ranking members of the Central and State Governments was focussed to several issues connected with the medical and public health problems of the country as a whole and of the Madras State in particular.

The Union Finance Minister inaugurated a new experiment at the Andhra Mahila Sabha's nursing home on the 17th August 1952, when he stressed the need to utilise the capacity of Indian women for social work in larger measure. He gave also wise counsel while addressing members of the various other maternity and social welfare and uplift centres he visited in Madras. The Governor of Madras, the Health Minister of the Madras State and the Vice-Chancellor of the Madras University had all very valuable advice to give to the pædiatricians of India who met in conference on the 24th August '52. The Union Minister for Commerce and Industry expressed on 3-9-'52 his desire to investigate the pressing problem of medicinal drugs manufactured in and out of India.

Inaugurating a three-day conference of District Medical Officers and Superintendents of State Hospitals in Madras on the 22nd August at the Madras Secretariat, Shri C. RAJAGOPALACHARIAR, Chief Minister covered a wide range of topics relating to medicine and public health in all their varied aspects—scientific, financial, administrative and social. He considered that such periodical meetings between the Ministers of State and the Chief Medical Officers in the Districts and

City Hospitals, augured well for the future and would create a better mutual understanding and appreciation of the administrative problems. He was of the opinion that women were more suited for the medical profession. It would be good for mankind if women picked and chose lines of human service and social service such as medicine, for which they were specially endowed by nature. He felt that medical administration at the top had now become more administrative than medical or scientific and that the services of competent surgeons and physicians were lost to society when they were appointed to administrative posts. Stating that they had however, gone too far now in departmentalisation the Chief Minister emphasised the need for greater co-ordination in the districts. The Community Projects which are now before the country are meant to co-ordinate public service in all its branches, instead of the various services splitting away and remaining un-co-ordinated. RAJAJI therefore, enjoined all medical officers in the districts to co-operate with the Officers-in-charge of these Projects and with the Collectors of the respective districts, without feeling piqued or offended by the demands made from time to time on their attention and valuable advice by these officers.

After discussing the various systems of medicine RAJAJI stated that the practitioner of the indigenous system of medicine firmly believed that the human body is one and the various organs of the body are not separate, and therefore, the treatment should be unitary. "From this fundamental article of faith modern medicine, had strayed away for a time" said he, "but I feel you are coming back again to the unitary doctrine of the human body and the treatment of human ailments and to the view that you should drive a many-pronged attack on any disease you find in the body so that the human system as a whole co-operates and brings about the cure of the conditions you find".

The Chief Minister appealed to the officers to take into account their needs and resources and arrive at priorities so essential in all planning, based on a correct appraisement of their resources, obligations and commitments.

Shri A. B. SHETTY, the State Minister of Health explained the difficulties in the way of development and improvements needed in several directions in medical service. The lack of funds and the lack of trained personnel were the main obstacles. He referred to the need for improving the quality of anæsthetic and out-patient work. He considered it necessary that medical graduates should have at least 5 years' training in large hospitals, before they can be put in independent charge of hospitals. Ampler facilities were necessary for post-graduate training ; with the upgrading of selected departments of hospitals in Madras city, Vellore and some other places and with the establishment of the All-India Medical Institute at Delhi, there

will be opportunities for the training of some more medical officers. The Minister stated that the working of the Honorary system required to be reviewed, in order to make it more useful and popular, and that Government in the Public Health Department was now considering a proposal for instituting refresher courses in important district hospitals. He was not unaware of the existence of many defects in the out-patient departments of all hospitals, the more important of which were want of adequate waiting accommodation, long hours of waiting before patients are seen by the staff, and perfunctory examination of patients. From the statistics published by the State Medical department in its annual administration report for 1950—the latest before us—it is seen that on an average one hundred and twenty patients have to be examined every morning in each of the 1229 Medical Institutions in this State. It is needless to say that nothing more than perfunctory attention is possible because 120 or more patients have to be disposed of in 180 minutes or less ! !

Apart from the above statistical considerations, those who have visited the large hospitals will know how the crowds of sick and suffering patients struggle for admission to the out-patient departments; most of these hospitals are also understaffed. Several patients coming from remote villages fail to reach the dispensary in the nearest town before the closing hours and have to wait till the next day or return disappointed. As pointed out in our last issue of the ANTISEPTIC the out-patient departments of all hospitals should be kept open for 3 hours in the morning and for at least 2 hours in the evening; and they should be provided with a larger staff in order to be able to meet the demands made on them.

A growing menace to health is the blatant sale of spurious drugs, inspite of the existence of the Drugs Act of 1940 and the rules made thereunder. This matter also came up for discussion at the conference and a recommendation was made to Government that steps should be taken to ensure the proper standard and purity of the drugs. Complaints are often heard about the sufferings caused by the administration to patients, of drugs which are below the proper standard or are short of the ingredients they ought to contain. But such cases are often ignored by the medical officers as being mere accidents, and no action is taken. The medical officers should promptly and without hesitation report such cases to Government. The supply of medicines manufactured by reputed firms which bear their seal and specifications in clear terms should *alone* be encouraged and allowed to be sold.

The shortage and consequent high prices of effective and reliable products are probably responsible to some extent at any rate, for the prevailing widespread entry and sale in the open market of spurious, useless or even harmful imitations. Where drugs of the specified quality and standard are in short production

or supply in India, it is necessary to obtain permits for imports. Import duties on such *useful, efficient, standard remedies not yet manufactured in our country* should be considerably reduced from their present high range of between 24 to 37½ per cent of the value of the medicines. To prevent the malpractices that prevail, a tightening up of the Drugs Act, is very urgent by amending the rules so as to penalise very heavily—even by prescribing imprisonment for those who resort to devious, dangerous and inhuman methods of getting rich overnight. The drug industry in India is in its infant stages and in its own interests, should enlist Government's co-operation and legislative action to eliminate the black marketers and unscrupulous nostrum makers and sellers. The State Governments should also be very watchful and vigilant through their medical officers in bringing the offenders promptly to book and thereby eradicating what promises to become a very serious menace to health than even epidemic diseases. In this connection we would also refer to the speech of Shri T. T. KRISHNAMACHARI the Union Minister for Commerce and Industry while addressing the Chemists and Druggists Association of Madras, on the 3rd instant; he was emphatic about the necessity for prompt action in regard to the control and stabilisation of the drug trade in India. He was however, referring chiefly to high cost of drugs manufactured out of India. He proposed to institute an enquiry with the concurrence of the Union Health Minister whether the drugs manufactured in India were being properly made and if they conformed to standards; that indeed is the right attitude to adopt, but until it is definitely known that everything at home is quite all right it would not be wise or proper to ban imports of drugs manufactured out of India. The Commerce Minister was right in wishing to have the advice of eminent physicians as to the lines on which the investigation should be made and decisions reached. His Madras speech will, we are sure, serve to open the eyes of foreign manufacturers to the urgent necessity of their reducing the phenomenally high prices they have been charging hitherto.

On the subject of Rural Medical Relief, the conference considered the institution of mobile dispensaries attached to base hospitals and the training of more medical men by means of a shorter and less expensive training course as the two solutions now commonly proposed. The Health Minister asked for the co-operation between the staff of the hospitals and general practitioners in the districts and emphasised also the need for the establishment of public relations by inviting the public at intervals to socials, showing them round the hospitals and discussing their needs.

The conference then proceeded to discuss various problems relating to the hospitals *viz.*, Honorary Medical Officers, appointments of specialists in various departments, hours of attendance etc. Suggestions are reported to have been made to the effect that

in every large hospital, the senior officer should attend to the new out-patients and the juniors to the old cases; also that if the District Medical Officer or the senior officer of a hospital was an able *surgeon*, his chief assistant should be recruited from amongst the best *physicians* and *vice versa*. Both of these are admirable suggestions and we strongly commend them to the notice and acceptance of Government for being promptly put into practice.

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# THE TRAINING OF NURSES

## A NEW EXPERIMENT IN MADRAS

DECLARING open the Andhra Mahila Sabha Nursing Home and Training Centre for nurses at Adyar Bridge Road, Madras on Sunday the 17th August 1952, Shri C. D. DESHMUKH, Union Finance Minister stated that the Planning Commission envisaged the formulation of a scheme to utilise the capacity of the Indian women for social work in a larger measure. Referring to the great shortage of nurses in hospitals, the Union Finance Minister furnished statistics to show that the ratio of nurses to the population was one to every 220 in Canada, one to 350 in the United Kingdom and one to 520 in the United States of America and one to 35,000 in India! In the Madras State which was as usual ahead of all other States in India, the ratio of trained nurses to the population was *one to 8,000* Even on this latter basis, it would take 100 years to reach the target of 7,40,000 nurses that will be required for the entire population of India as it stands to-day. There were really three difficulties in the way of getting a large number of nurses:—(1) the lack of young women with the requisite qualifications willing to enter the profession; (2) the dearth of properly equipped and adequately staffed training institutions; and (3) the perennial problem of want of funds.

In order to solve the problem of shortage of nurses a scheme approved by the Indian Nursing Council aimed at giving a short course of training to prepare auxiliary nurses to work in Institutions or in the homes of patients. "This" said Shri DESHMUKH "might be claimed as the first attempt at simplifying the problem and providing nursing service to a little larger section of the population than at present. This new experiment launched by the Andhra Mahila Sabha, was really of great consequence from the planning point of view. It is the first scheme the Union Government has allowed a private organization to undertake". The Madras State Health Minister Shri A. B. SHETTY agreed that there was great need for the expansion of nursing and midwifery. "Malabar" he said "contributed the largest number of women

for training in nursing and midwifery. Next came the Tamil Districts and the Andhra Districts sent comparatively few women." He was therefore, particularly glad to note that the Andhra Mahila Sabha was adopting this shorter course of training. Dr. A. LAKSHMANASWAMI MUDALIAR, the Vice-Chancellor of the Madras University welcomed the scheme and said "it would help them to train a large number of midwives who would be in a position to render maternity help in rural areas which needed considerably more attention".

We quite appreciate the great and good selfless work of Shrimathi DURGHABAI, to whose zeal and unflinching efforts and persuasive advocacy, is due the present new venture so eloquently blessed by our Union Finance Minister and those who spoke after him. But neither Shri DESHMUKH nor the others placed in authority, indicated where Governments and local bodies were going to get the funds which will provide employment when the period of training was completed. It is not as if they may be asked to go and settle *on their own* in rural areas and earn their livelihoods by private practice; nor would small subsidies, even which can only be grudgingly made by local bodies, help these messengers of hope and healing to live in peace and comfort. The Planning Commission has also blessed the new training centre and the Union Government has kindly sanctioned a non-recurring and also a recurring grant for 5 years in the first instance, for running this centre. The nurses to be trained here, it is stated, will take a shorter course that will enable them to take up the work of maternity relief in *rural areas* earlier than if they had to take the full course of training for nurses We do realize the great need for many more thousands of nurses for our country, and also the great scope that exists for a close co-operation between the Centre, State Governments and voluntary organizations in affording facilities for *suitable and adequate* training for them. While the minimum educational qualification for a full course of training (3 years) in nursing is the possession of a S. S. L. Certificate, the minimum asked for in respect of this new shortened course of 2 years, is the seventh standard or even a special entrance examination, presumably by the admitting authorities. These nurses trained under this new scheme may not be able to deal with complex cases, though they may be all right for simple minor ailments. Even in the matter of their training the lower level of general education may and perhaps will in most cases, prove a serious handicap to a quick grasp and a proper appreciation of the different advanced methods and techniques of sick nursing.

To our mind the training of nurses, particularly of those who have to work in rural areas should be very thorough and complete as they will frequently be called upon to work independently and cope with emergencies, without a doctor's help being available near at hand.

The best place therefore, to give training is in the large and fully equipped hospitals maintained by the State in the City and in the head-quarters of the districts; here the trainees will find facilities for learning to solve many difficult points which may arise in the course of the day to day work which later, they will be called upon to tackle. In his address of welcome to the pædiatricians of India who were in conference at the Madras Medical College on 24-8-'52 our worthy Vice-Chancellor Dr. A. L. MUDALIAR said "The importance of the nurse in pædiatrics cannot be overlooked and the time is come when there should be a definite period of training in a children's clinic for all those who are undergoing general training in nursing while the pædiatric nurse and the public health nurse should have a more specialised form of training which will include domiciliary visits and training in the preventive and curative aspects of medicine." Coming from an experienced and eminent top-ranking obstetrician *cum* pædiatrician of India, this advice is entitled to be heeded and followed. In the republican-set-up we have in India today, every child, whether in city, town or village is important, as the Governor Shri SRI PRAKASA said, because when grown up, he might be called upon to shoulder the most difficult of responsibilities almost at a moment's notice. And should the children in the rural areas not therefore, receive the benefit of the care, advice and attention of the *fully qualified nurse* with some training in pædiatrics also ?

Shrimathi Dr. MUTHULAKSHMI REDDI pointed out at this very meeting that the bulk of the medical and nursing professions in the Madras State was concentrated in the urban areas, only 7 per cent of the child-births in the rural areas of the State getting skilled maternity help, though the large majority of the population of the country lived and toiled in the rural areas with few or no amenities. There is thus a most pressing need for increasing the number of *fully qualified nurses* and making them available for service to the larger rural population. The issue is not at present merely one of training only. It is also perhaps one of finance—the offering of sufficient inducement to educated young women to take to nursing as an honourable profession of service to humanity and also making the amenities and emoluments for them attractive and adequate after the training is finished and when they are detailed for work in rural areas.

The Union Finance Minister paid a well-merited encomium to Madras as being ahead of other States, even in the matter of having more nurses and affording them greater facilities and amenities of life. It is no doubt true that nurses in Madras are perhaps somewhat better-off than in other States in the matter of accommodation and amenities, but the ideal is far from being achieved yet. There has been a welcome change in the outlook and also a better understanding by the public of what "nursing" really means and how it

is more important than even doctoring in illnesses. Nearly all hospitals are still inadequately manned as regard qualified nurses and the public who prefer to be treated in their homes when they can afford it, are unable to get the services of qualified nurses when they need them badly. Many of the larger towns in the moffussil are without this amenity of having qualified sick nurses in private practice; while the hospitals themselves are badly in need of these 'ladies with the lamps' how can the public expect to have their services for domiciliary treatment till we have quite an army of them ?

The Union Finance Minister, admitted the fact of poor and inadequate allotments for Health and Education and after he had seen for himself the wonderful work carried out in the many social and welfare institutions he saw in his round of visits in Madras, he is reported to have kindly promised to afford some relief. We trust he will do so and persuade his colleagues at the Centre that Madras which has been leading in all directions deserves special consideration and greater encouragement by the diversion of larger sums of money for the expansion of medical relief in general and of this branch of humane endeavour in particular.

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### *Ætiology of Diabetes Mellitus : The Changing Concept*

The ætiology of diabetes mellitus presents a number of different and complex problems. Apart from its remarkably great therapeutic value in clinical medicine, the isolation of insulin stimulated a large volume of clinical, biochemical and experimental work, with the result that medical opinion is now receding away from Allen's concept of the primarily pancreatic origin of diabetes and even from the hitherto generally accepted idea that it is a single disease-entity. Autopsy material collected from patients who have died from diabetes, has failed to reveal characteristic changes in the islet cells, in many cases. Diabetes mellitus seldom occurs either during the course of or as a sequel to acute pancreatitis. Assays of the insulin content of the pancreas from diabetics have on occasion yielded quite normal values. Downie, is of the opinion that *the experience of 30 years has swung medical thought and opinion away from the view that diabetes mellitus is caused by a primary pancreatic deficiency.* The one essential feature of diabetes mellitus is hyperglycæmia, which may arise, in some instances atleast, from increased utilization, damage, or destruction or inhibition of insulin rather than from a primary decrease in the production of that hormone. A great deal, no doubt remains to be discovered by further sustained observations and research, but *at present diabetes mellitus is better regarded as a symptom complex resulting from any of several causes than as a disease, sui generis.*—(*Med. Jour.*, Austral., 1 : pp. 110-112, 1951).

## MEDICINE AND THERAPEUTICS

**Sulphonamides and antibiotics in amœbic dysentery.**—Dr. S. J. Das Gupta, M.S.C., D. Phil., Sir Profulla Chandra Research Laboratory.

It is now generally agreed that neither penicillin nor sulphonamide has any direct action on E. histolytica. Nevertheless there is considerable evidence to show that by their action either on intercurrent bacterial infection of the intestinal wall or on the bacterial flora in the intestine, these compounds may bring about clinical improvement in cases of long standing amœbic dysentery. The importance of bacterial flora in amœbic dysentery has been emphasised by Acton and Knowles (1928) and it is no wonder, the value of penicillin in chronic amœbiasis has since been confirmed by many workers (Blanc and Siguier 1946; Benhamou *et al* 1947). Sulphaguanidine, sulphasuxidine (Succinyl-sulpha-thiazole) and a few other sulpha drugs have for similar reasons been recommended in both acute and chronic cases of amœbic dysentery. In fact, the removal of secondary bacterial infection from the intestinal wall and the possible alteration of the bacterial flora of the intestine by antibiotics and sulpha drugs mark a definite advance in the chemotherapy of amœbiasis.

Extensive clinical trials are being conducted throughout the world with the newly discovered antibiotics such as aureomycin, bacitracin, chloromycetin and terramycin in amœbiasis either alone or in combination with other amœbicidal drugs. Of these antibiotics, aureomycin has been found to give comparatively more convincing results It was shown by Hewit *et al* (1950) that aureomycin had a marked amœbicidal effect in a dilution of 1 in 1000. Similar tests *in vitro* were made with 1 in 100 of chloromycetin, streptomycin and bacitracin, but none of these antibiotics had any measurable effect on E. histolytica.

Most *et al* (1950) treated 32 cases of amœbiasis with aureomycin 1 to 2 g. daily in divided doses for 10 days. In all the cases except one, amœbæ disappeared from the stools. Toxic symptoms such as vomiting and diarrhœa were observed. Siguier *et al* (1950) treated 40 cases of amœbic dysentery with aureomycin, 20 to 30 g. of the antibiotic given orally during 7 to 10 days; 1·5 to 2 g. was given on the first day and 3 g. on the following days. The drug was well tolerated, only 4 patients showing minor disturbances. The action of the drug was rapid. In 35 cases a clinical cure was observed.

Mcvay *et al* (1950) treated 37 cases of amœbiasis with 0·5 g. of aureomycin four times daily. The drug was well tolerated, gave rapid relief in severe cases and was effective in 36 out of 37 in eliminating cysts and vegetative forms from the stools.

Armstrong *et al* (1950) treated 52 cases of amœbiasis with aureomycin given orally in doses of 0·25 g. four times daily at 6 hour intervals for 15 days. The results compared favourably with other forms of treatment but they suggested that the antibiotic may be combined with other amœbicidal agents.—(*Advance Therapy*, July–Dec. 1951).

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**Resochin in amœbic hepatitis.**—(Subramaniam, R., *Ind. Med. Gaz.*, July 1952).

Reviewing a series of cases of amœbic hepatitis treated with resochin, Dr. R. Subramaniam has given details of the clinical efficacy of the drug. The composition of the drug is 7 chloro-4- (4 diethyl-aminol-methyl-butylamine) quinoline diphosphate. It is also known as chloroquin. Originally used principally as an antimalarial, it has now been found effective also in cases of amœbic hepatitis. It is rapidly absorbed from the intestines and stored in tissues of the body especially the liver. Cases of amœbic hepatitis as well as of abscesses of the liver were included in this trial. The

response was demonstrated by a fall in temperature, relaxation of guarding of right upper abdominal quadrant, improvement in the general condition of the patient and decrease in tenderness of the liver. The response was better in cases of hepatitis than in cases of abscesses. But when the abscess was aspirated and the patient then put on resochin, the relief was marked. The dosage recommended in all cases was as follows: For the first 4 days one tablet every 6 hours. From the 5th to the 12th day one tablet was given twice daily and from the 13th to 21st day one tablet once daily. On an average between 37 to 44 tablets would be required for a full course. The tablets are to be administered after food. The advantages of resochin over other amœbicidal drugs were:—

(1) The relative non-toxicity as compared with emetine etc.

(2) The quicker relief of pain in cases where the pain was very acute.

(3) The quicker healing of pneumonitis which often occurs as a complication of hepatitis. This was particularly noticeable when, as a result of the healing of the lung lesion with resochin, the movement of the right dome of the diaphragm became freer than before.

Resochin therefore, marks a definite advance in the treatment of amœbic infections of the liver.

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**Allergy to aspirin.**—(*Canad. Med. Assoc. Jour.*, 64: pp. 187-192, 1951).

The drug giving rise to allergic reactions most frequently is acetylsalicylic acid (aspirin). The most common symptom is asthma; but angioneurotic œdema, urticaria and rhinitis are also fairly common. Definite sensitivity to acetylsalicylic acid was noted by Walton and Bottomley in 22 of 830 patients with asthma. All the twenty two patients promptly manifested asthma when given a small dose of aspirin. Several of them had urticaria, rhinitis and angioneurotic œdema. Three of them were found to be also allergic to antihistamine. Seven of the 22 (all adults between the ages of 27 and 64) were men and the rest women. While only 108 of the total 830 asthmatics (13%) suffered from a severe type, eleven (50%) of the 22 (aspirin—allergic) patients had a severe degree of asthma. Five of these twenty two died in the four year period covered by this study. Necropsy was performed in 3 of these 4, and all the three showed the characteristic changes of fatal bronchial asthma. Clinical reactions to acetylsalicylic acid are usually violent and rapid.

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## SURGERY

**Re-establishing lymph drainage in lymphœdema.**—(*Jour. Internat. Coll. Surg.*, Chicago, 15, pp. 328-331, 1952).

Dr. Zieman reports rapid and painless reduction of the affected part by a simple, non-mutilating method of re-establishing lymph drainage in an extremity subject to lymphœdema. His technique consists of subcutaneous implanting of one or more lengths of non-absorbable suture longitudinally along the extremity and across the region of the obstruction to the lymphatic flow. A practically new channel is thus created, permitting the drainage of lymph. The suture material (Nylon No. 1) is introduced through the bore of a No. 19 or No. 20 gauge needle 17·7 to 30·4 cm in length, that has previously been inserted into the skin (subcutaneously) along the required path. The needle is withdrawn leaving the suture alone in place. The ends of the suture are cut, so that they retract 0·6 cm. under the surface of the skin. Immediately after the above implantation, the extremity is elevated and wrapped up in an elastic bandage. Zieman states that he has used this method successfully in lymphœdema of the arm following radical mastectomy, lymphœdema of the leg due to invasion of the inguinal nodes by a Hodgkin's infiltrate, and in lymphœdema of the penis and scrotum and every case resulted in quick painless reduction of the affected member.

**Surgical management of secondary hypersplenism.**—(*Surgery*, St. Louis, 29, pp. 407–408, March 1952).

Two notable examples of hypersplenism are idiopathic thrombopenic purpura and congenital icterus. Lord (Jr.) presents five illustrative cases of hypersplenism. Splenectomy benefits both conditions usually. In secondary hypersplenism, the following signs and symptoms are seen:—(1) enlargement of spleen; (2) bone marrow is normally active or hyperactive; (3) there is evidence in the peripheral blood of a reduction below normal of all or some of the cellular elements; and (4) there is some primary disease condition, involving the spleen directly or indirectly. Blakemore *et al* have developed satisfactory shunting procedures for the relief of portal hypertension on both an intra-hepatic and an extra-hepatic basis. In patients with secondary hypersplenism due to a congestive portal hypertension; combined splenectomy and spleno-renal shunt have proved very effective.

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**Control of bleeding in cranial operations.**—(*The Lancet*, 1-9-1951).

The control of hæmorrhage is a major problem to the neuro-surgeon. In cranial operations the blood-pressure can be reduced by various means, ranging from chloroform anæsthesia to placing the patient in the sitting position. These methods are not however, always reliable and they may even be dangerous.

*The newer methods of reducing the blood-pressure* during operation are arteriotomy, and the intravenous injection of methonium compounds. So far methonium compounds have been little used in surgery of the brain but arteriotomy has found enthusiastic supporters both in Europe and America. Blood is drained from the arterial circulation until the blood-pressure falls to the desired level; and the blood thus obtained is citrated and re-infused at the end of the operation. Many advantages are claimed for this procedure. It gives a bloodless field, as a result of low blood-pressure and reflex vasoconstriction. Furthermore, the cerebral cortex shrinks owing to the simultaneous lowering of the C.S.F. pressure; and access becomes correspondingly easier. Operation on vascular tumours and structures, is greatly simplified because of the lessened hæmorrhage; and lastly demands on the blood-bank are greatly reduced.

Like arteriotomy, the injection of methonium drugs produces a fall in both the blood-pressure and the C.S.F. pressure. Arteriotomy however, evokes pronounced compensatory peripheral blood-flow, though this may be masked by the low blood pressure. Arteriotomy might thus seem to be more suitable for controlling hæmorrhage. With it, the blood pressure can be quickly raised to normal, if this should prove necessary—for example to ensure perfect hæmostasis before closing the wound. Arteriotomy calls, however, for undivided and skilled attention and for special apparatus. It is unlikely therefore, to find wide adoption.

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**Rehabilitation after poliomyelitis.**—(*Pres. Med.*, 59 : 413-420, 1951, *Eng. Abst. W. M.*, 10 : 3 : 245, 1951).

Leroy and Ryckelyneck in rehabilitating patients suffering from poliomyelitis, used in conjunction with balneotherapy, mechanotherapy, by means of roller skates applied to the feet and hands. By this method they excluded the action of gravity and the forces of friction and inertia, which certainly interfere with the proper exercising of weak muscles. The roller skates by means of a simple adaption can be attached to the limb so as to allow movements in every direction.

A wooden board 2 metres (6½ feet) long and 1 metre (3½ feet) broad and inclined at an angle of 30 degrees so as to avoid friction of the leg against the bed is part of the equipment for exercising the muscles of the upper and lower limbs in bed-patients. Joint and muscle proprioceptive sensation is thus restored in normal surroundings and this is an advantage over balneotherapy where the patient is exercised in a medium in which he will not eventually live.

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## REVIEWS OF BOOKS, PERIODICALS AND REPORTS

**Bone Tumours**—[By LOUIS LICHTENSTEIN, M.D., Senior Pathologist, General Medical and Surgical Hospital, Veterans' Administration Centre, Los Angeles. pp. 315, with 155 illustrations. Louis: C. V. Mosby Company, 1952.]

We are indebted to American authors for very exhaustive information on bone tumours. "Tumours of Bone" published by Geschickter and Copeland, is perhaps the best reference book we have had from that great country. The addition of yet another monograph on the same subject by Lichtenstein is most welcome. The material he has published is most valuable as it is based on personal observations, during his long and intimate connection with the Hospital, for Joint-Diseases in New York. The author has covered all aspects of the subject admirably but could well have omitted the large volume of historical, anatomical and developmental aspects that could be had from other books. The reader cannot fail to appreciate the impartial manner in which he has discussed some of the controversial subjects, like giant cell tumour and Ewing's sarcoma. The get-up of the book and the illustrations are all excellent. The surgeon, pathologist and radiologist will find this book extremely helpful in solving some of he knotty problems on bone-tumours.

D.G.R.

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**Pædiatric Allergy**—By ROBERT CHOBOT, M.D., Assistant Clinical Professor of Pædiatrics, New York University, Bellevue Medical Centre, Director of the Pædiatric Allergy Clinic, University Hospital of New York University—Bellevue Medical Centre; Consultant in Allergy; Downtown Medical Centre; Assistant Physician, Allergy Clinic, Roosevelt Hospital. First edition 1951, 284 pp. Published by McGraw Hill Book Company, New York, 330, West 42nd Street.

*"Allergy" is a condition which is vaguely understood and is* very often loosely applied to conditions whose orgin is not very clear. Such, when found in children are still more complicated. The author who is a leading authority on the subject has attempted to simplify the whole matter and achieved a fair measure of success. The subject-matter has been divided into a number of convenient chapters. The first two deal with various forms of hypersensitivity as well as diagnostic procedures including various skin tests such as, the scratch test, the intradermal test, and the patch test. Asthma and food allergy are next dealt with. The chapter on asthma is very well written and great care has been bestowed in presenting the latest views on the subject. Hay fever, a condition which is not common in our country is next described in great detail as it is very common in western countries; this chapter has been illustrated by charts showing the causative factors operating in different parts of the United States. Skin conditions which have an allergic back-ground are next described. Eczema, which is resistant to usual methods of treatment has been well discussed as also the modern methods of treatment. Serum reactions and drug allergy, allergy of the eye and migraine have been dealt with in separate chapters. Brief reference has also been made to the present-day use of ACTH and cortisone in pædiatric practice. The last section contains an appendix with a series of cases of pædiatric allergy and the investigations made in such cases. This is an outstanding feature of this book.

The book has been properly indexed and is printed in clear and bold type. It should be useful to the pædiatrician and also to the general practitioner commanding a large pædiatric practice.

U. V. B.

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## ACKNOWLEDGEMENT

We have received a copy of the August '52 issue of the Indian Medical Practitioner, Bahadurgarh Road, Delhi. It deals largely with Ayurveda.

## CORRESPONDENCE

To The Editor, ANTISEPTIC, Madras.

### Modern Treatment of Malaria

Sir, I was much interested in the article on Modern Treatment of Malaria by K. N. Sachdeo (August, '52 p. 606). Writing about chloroquine, the author observes that it is available in tablet form as the diphosphate, each tablet containing 0·25 gm. of the base. This statement needs amending because chloroquine is available in tablet form both as the diphosphate and the sulphate. As the diphosphate, chloroquine is presented in tablets of 0·25 gm. of the salt containing 0·15 gm. of chloroquine base and not 0·25 gm. of the base. As the sulphate, it is presented in tablets of 0·2 gm. containing 0·15 gm. of chloroquine base.

The author further observes that chloroquine is thought to be equivalent to Proguanil in its antimalarial activity Whether it is the mode of action or the effectiveness of the drug in the therapy and prophylaxis of malaria to which Dr. Sachdeo is referring, this does not represent the true status of chloroquine. About the comparative effectiveness of the various antimalarials in the therapy and prophylaxis of malaria, the following statements on chloroquine from the statement on Modern antimalarials by the Expert Committee on Malaria of the World Health Organization (W.H.O. Tech. Report Series No. 8, May 1950, p. 7) and the report of the Scientific Advisory Board on chemotherapy of malaria based on an enquiry by Dr. R. N. Chaudhuri at the School of Tropical Medicine, Calcutta will be found interesting.

"Recent studies have shown that chloroquine is effective when given parenterally, and as a suppressant its action is unsurpassed by any other drug against all strains of malarial parasites so far tested. P. vivax was completely suppressed when 0·5 gm. of chloroquine was given once weekly for 6 months to one group of experimental subjects and for one year to another group. It has been shown that 0·5 gm. once a week gives almost complete protection against the three common forms of human malaria. For children a dose of 0·25 gm. was found to be effective and was well tolerated."

"Other drugs closely related to chloroquine have similar antimalarial properties. Of these sontochin, oxychloroquine and camoquin are the most effective and are relatively non-toxic. None of them, however, is superior to chloroquine." (W.H.O. Tech. Series No. 8.)

"Clinical cure was effected in all the cases; the asexual parasites disappeared from the peripheral blood within three days after the treatment was started in 80 per cent of those treated with paludrine and 100 per cent of those who were given chloroquine and camoquin and the fever was controlled in 84 per cent, 100 per cent and 100 per cent of the cases, respectively. All the chloroquine and 80 percent of camoquin cases became parasite-free by the end of the second day, and 84 per cent of the former and 76 per cent of the latter became afebrile. Thus, all the three drugs are effective in bringing about a clinical cure, chloroquine being quick, paludrine slow and camoquin intermediate."—(*Report of the Scientific Advisory Board* for 1949).

Madras,
1st Sept. '52. } G. G. KRISHNAMURTHY.

[Dr. Sachdeo has been contacted in the matter and his opinion will be published as soon as it is available.—ED.]

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To The Editor, ANTISEPTIC, Madras.

### Local Fund and Rural Practitioners

Sir, A Conference of the District Medical Officers is going to be held by the State Minister for Health very shortly to consider the question "How to increase the efficiency of medical aid in the State and especially in rural parts". The medical aid to the rural population is now provided partly by the State Government and partly by local bodies. Qualified and energetic medical men find that the terms offered are not attractive enough to serve in the institutions run by the local boards, and it is found very difficult to find suitable men to be employed in these institutions. Therefore, only old and

retired people are now employed for these institutions; where even such old and retired people are not to be had, some dispensaries are run without doctors and others have been closed. Government knowing the cause for this state of affairs, have not so far come forward to remedy this. There is a wide disparity in the pay and status between the men in government service and those employed by the local bodies, with the same qualification and for the same kind of work. The maximum pay of the latter class is the starting pay of the Government man—or even less. Further the men in L. F. service have no opportunities to improve their knowledge of medicine that the men in government service have, nor have they the chances of getting additional income from private practice, as all the rural areas are very impoverished. Those few stations that are attractive and offer some decent practice, are given only to the favourites. Thus, the others are to remain content with any out-of-the-way station to which they may be posted without any consideration for their seniority or merit. They have to get on with the meagre pay in these hard and difficult days. While such is the condition of the medical men in the Local Fund service, how can the public expect efficient medical aid and enthusiastic service?

I hope and pray that the Government will come forward with their remedial measures before accusing medical men for not choosing to serve in rural parts.

13-8-1952 "MEDICO"

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To The Editor, ANTISEPTIC, Madras

## The Doctor and His Family

Sir, I enjoyed reading the article "The Doctor and His Family" by a very distinguished writer in the current issue of our journal. It is very true that doctors are as human as others, and are unaware that they are equally handicapped by ignorance, superstition and prejudice, though of course, these appear in different forms. Regarding the example of the science student cited, I wonder if it was a case of prejudice, or if he had a visual defect which made him see the colour of chromium as normal eyes did not.

It is refreshing to note the emphasis on clinical examination of the chest which seems to be a lost art in these days. The clinical findings formulate the diagnosis, which is then confirmed by laboratory or X-ray investigation. Perhaps one may be permitted to emphasise that these investigations should also be done when clinical findings justify the need for them.

While it is undoubtedly true that doctors are handicapped by prejudice in the case of illness amongst their relatives, I wonder how many will agree with the cause given. Perhaps there are "high born" doctors who consider certain diseases as "vulgar" and affecting only the "lower" strata of society. There are probably others (I should think a good number) who, when confronted by illness in their relatives, unconsciously keep out of their minds diseases which they dread, as a result of ideas gained either during their training, or experience as doctors. I know of some others too, to whom every case of tonsillitis in their children appears to be diphtheria, and even the mildest fever a case of typhoid. Jaundice occurring in an elderly member makes them suspect cancer of the liver and pancreas. Each of the above group of doctors is affected by prejudice, but the causes and effects are different. This prejudice acts not only in the case of relatives but also of close friends. The remedy is simple, but unfortunately, not often used; namely, to resolve not to treat one's own relatives.

Lastly, the aptness of the remark that "It is the habit of Allopathic doctors to say that nothing need be done in the earlier stages of a malignant disease....." is open to question. This is the age when surgery is regarded as an inconvenience rather than a terror, and "prophylactic" (if one may use the word) removal of benign tumours, diseased uterus and other organs is advocated and practised on an increasing scale. The whole fight against malignant disease at present is based on taking radical measures at the earliest.

Anaimalai, 4—8—'52. K. N. RAMAN.

# NEWS AND NOTES

## Powerful New Drug Fights Malaria

A new weapon has been found in the fight against malaria, which kills more than 3 million people a year and affects some 700 millions.

Daraprim, as this new discovery is known to the medical profession, was developed in the laboratories of the Wellcome Foundation in London and New York.

Its outstanding characteristics are that it is 1,000 times as powerful as quinine, is absolutely tasteless, can be taken in tiny doses, and does not cause toxic after-effects such as sickness or depression.

Tests have been conducted in India Tunisia, Indo-China, the Belgian Congo, and West Africa and further tests on a larger scale are now being conducted in India. Daraprim is now available for general use.

In order to prove the efficacy of Daraprim, 36-year-old Dr. L. G. Goodwin, of the Wellcome Laboratories of Tropical Medicine while in Africa during the first half of last year, took daily doses of the new drug and deliberately exposed himself to infection from malaria, but he did not contract malaria. Still continuing to take regular doses of Daraprim, he allowed himself to be bitten by four anopheline mosquitoes which were dissected and found to be carrying malaria, yet he remained free from attack. Daraprim at a dose of 5 mg. daily is a satisfactory suppressant of malaria and produces no ill-effects of any kind when taken continuously for a year.

—

## British Schering's Gift to India Government

The following is a cut-out from the '*Times of India*,' Bombay, dated the 5th August 1952:

Messrs British Schering Limited, London, have made a donation of 50 boxes of Liviron ampoules and 300 bottles of acetocid eye-drops to the Government of India for distribution in India. These are being distributed to the states of Uttar Pradesh, Himachal Pradesh, the Punjab and Saurashtra.

## Mepacrine helps relieve rheumatic pain

Mepacrine, which played a leading part in the Middle and Far East as a substitute for quinine in fighting malaria, may prove to be effective for the relief, of rheumatoid arthritis Freedman and Bach, in a preliminary report in the *Lancet* on the treatment of rheumatoid arthritis with Mepacrine state that all but one of 23 patients who had the disease for 2 years, showed considerable improvement.

Six patients who had cortisone were given Mepacrine instead and four of them were now almost free of pain. Three cases had a long course of gold without benefit, but the swelling and tenderness of joints subsided completely while they were having Mepacrine. Apart from physiotherapy 14 patients had been having salicylates only; when they commenced taking Mepacrine their pain gradually eased as their skin became yellow. Soft tissue swellings subsided and movement became free and more vigorous. Power of grip and speed of walking gradually improved.

These instances of treatment were however, uncontrolled and so it is too early to be certain, that Mepacrine may have been the effective agent.

—

## Trachoma Treated with Terramycin

The U. S. Army, in co-operation with American and Japanese scientists is conducting a programme on Okinawa against the major eye malady, trachoma. The project involves the use of the antibiotic terramycin in the form of ointment. Trachoma, an eye-crippling infection which can result in blindness, is a major health problem on Okinawa, as well as in Japan, India and other Far Eastern and Middle Eastern countries. In a survey of 4,017 persons conducted on Okinawa recently 453, or more than 10 per cent were found to be infected.

Terramycin's effectiveness against this eye disease was brought dramatically to light last year by Dr. Mitsui in clinical tests involving more than 700 trachoma

patients. An associate of his, Dr. Chie Tanaka, is working with American scientists on the current Okinawa project. (*M.P.I.B.* release, May 1952).

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### World Cancer Conference

An International Cancer Conference is to be held in Bombay in 1952. According to Dr. E. V. Cowdry the American Cancer Expert, working at the Indian Cancer Research Centre, Bombay. Many countries including Korea, Indonesia S. Africa, Britain, France and Belgium will participate in this conference.

A non-recurring grant of Rs. 30,000 was recently sanctioned by the Union Government to the Indian Cancer Society, Bombay for meeting part of the expenses in connection with the meeting of the International Cancer Research Commission to be held in Bombay in Dece. 1952.

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### The 29th All India Medical Conference

The Twentyninth All India Medical Conference will be held in Patna in the last week of Dec. 1952 along with the All India Ear, Nose and Throat, the All India Cardiologists and Pædiatrics Conferences.

### Filarial Research in India

The Union Government have decided to open a Filariasis Research section in the Malaria Institute of India, at Delhi for the study of the epidemiology of filariasis in India. The Indian Council of Medical Research had been agitating for this for a long time and has succeeded now in obtaining a grant of Rs. 40,000 for this purpose.—(*P.I.B. Bulletin.*)

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### International Medical Congress in London

Doctors from India, Pakistan and Ceylon are among those expected to attend the four-day International Medical Congress opening in London on September 15. The programme consists of four symposia, with speakers chosen from various countries and with special knowledge of certain aspects of the subjects under discussions. These include discussion on antibiotics in man, to be opened by Sir Alexander Fleming, discoverer of Penicillin, and virus infections of the nervous system, to be opened by Professor Paul of Yale University. A day will be devoted to discussing the significance of the up-to-date studies of body chemistry and fluid balance.–(*B.I.S. Bulletin* 19-8-52.)

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## OBITUARY

### The late Dr. M. G. Kini

We regret to record the death on 22nd August 1952, at Bombay of Dr. M. G. Kini, after a heart attack at the early age of 57. He was an able surgeon, much respected and exceedingly popular in Madras. He was responsible for organising the Orthopædic Department in the General Hospital, Madras. He was one of the founder members and past-President of the Association of Surgeons in India. On his retirement from the Madras Medical Department, after a long and brilliant record of service, he went to Bombay at the express desire of the Society for the Rehabilitation of the Crippled Children, to organize the Children's Orthopædic Hospital in Bombay, in which attempt he succeeded gloriously. At the invitation of the Union Government he led a team of medical relief workers for the relief of persons suffering from poliomyelitis during a severe epidemic in the Nicobar Islands. He was keen on research and did not care for wealth.

He was a distinguished Fellow of the Royal College of Surgeons and also of the American College of Surgeons. He won the distinction of being elected to the Fellowship of the Royal Society of Edinburgh. He is survived by his wife, three sons and a daughter, besides a very large circle of friends, grateful patients and devoted students who bemoan his loss. We extend our sincere sympathies to the members of the bereaved family.

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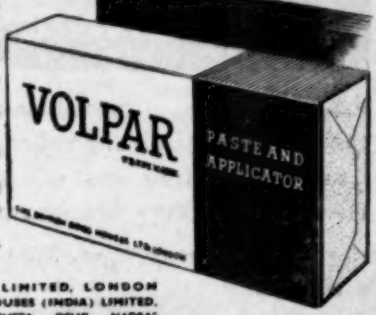


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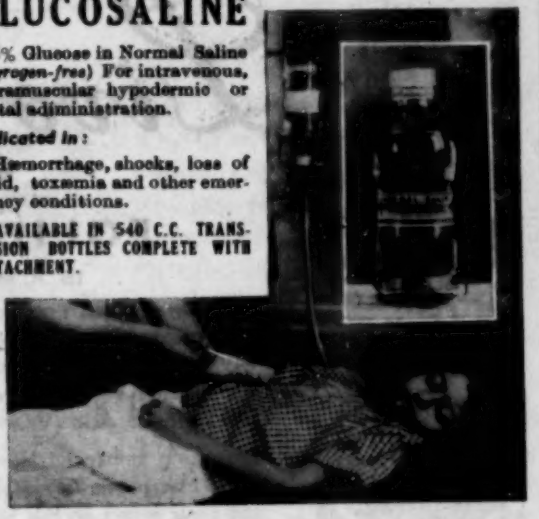

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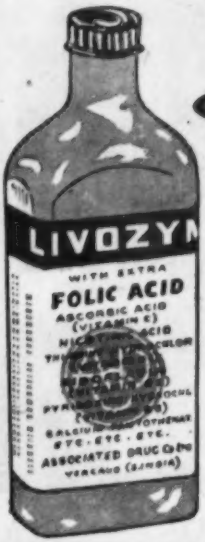


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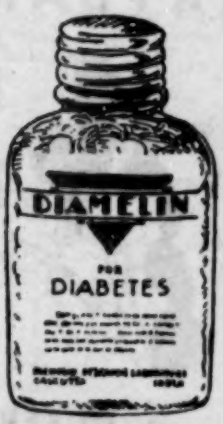


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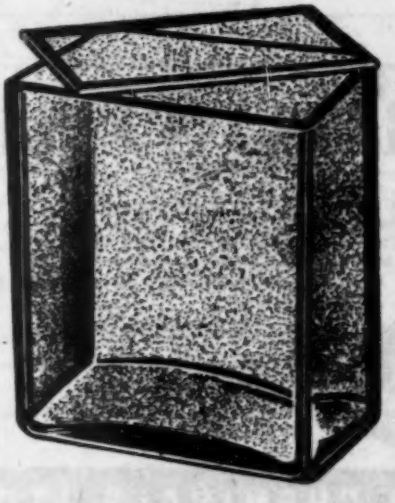

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4 lacs x 1-5-0 x
Dihydrostrepto. Pfizer 2-8-0
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" " " 12x1 lac 7-10
Glaxo " " 10x2 lac 5-8
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Lozengis 20 1-2
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" Liver Ext 10 cc. 2 USP 3-12
" " " 10 cc. 5 USP 7-12
" Adrenalin in oil 6x1 cc. 3-8
" Combex 10 cc. 6-14; Abdec
[drops 6-4
" Benadryl cream 2-7;
Pfizer Terramycin cap 8 17-0
[16 32-0
Lederle Aureomycin cap 8 18-14
Rhodia PAS tab 7½ gr. 100 4-0;
[500 17-4
PAC tab 75 6-2; 250 17-4
Dumex PAS 100 grm. 10-4;
[Italy 4-4
Quinine Sulph How oz. 3-12;
[lb. 51-0
" " Japan lb. 43-0
M.B. Santonine dr. 6-0
Eue Quinine Java 4-2; Roche
[oz. 4-8
How Qui Bisulph tab.
2 gr 100 2-0; 5 gr 3-12
" " Bihydro tab 2 gr 100
[3-0; 5 gr 5-0
Germ. " Bisulph tab 5 gr 1000
[33-0
BDH " " tab 5 gr 1000 37-8
Quinine amp 100x10 gr 2 cc
Ind. BDH Germ. P. Davis
17-12 23-8 23-4 39-8
11-8 16-4 100x5 gr 1 cc
Quinacrin tab USA 1000 12-4
Mepacrin tab 1000 10-8
Atebrin tab Bayer 15 0-10;
[300 7-14; 1000 16-8
Ephedrin tab ½ gr. 1000
" Merck 13-0; German 7-0
Paludrin tab 1 grm 1000 26-4
" 3 grm 125x8 Strepe 51-0
Sulpha tab 500 1000
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Sulphagunadine " " 12-8 24-8
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Strepto e PAS Leptit 5-4
Thermometer Zeal 2-7; USA
[1-6; Eng 1-8
" Japan 0-13; Jap. flat 1-12
Emetin Amp BDH 12x1 gr. 12-8
[12x½ gr 6-12
" " P.D. 6x1 gr 10-14;
[6x½ gr 6-12
" Endo USA 6x½ gr. 2-4
Cal. Gluconate amp 100x10%
[10 cc 13-8
Glue Sol 25% 25cc 100 amp 21-8
" " " Thille Germ. 50 20-0
" " " " " 12 5-8
Milk with Iodine amp 100x5 cc 16-0
Dist. Water amp 100x10 cc 7-12
[5 cc 5-12
Normal Saline 100x10 cc 8-8
[5 cc 6-12
Aspria tab 5gr 100 1-0; 1000 5-12
" Caffein tab 5 gr 1000 8-8
[A.P.C. 15
Vit B Comp 10 cc USA 4-10
Atophen amp IM/IV Germ. 5-10
" Tab 20 3-0; 100 11-6
Veramon tab 10 1-7; 20 2-10
Cylotropin amp IM or IV 4-12
Aletris Cordial Eng. 4-4
Sulphur Bitter Eng. 4-8
Glaxo Seclopen 20 lac 4-14
AFD Felsol pow 2-12; Bepiex tab 25
" Sulpharsenol [1-13
1 2 3 4 5 6
1-1 1-3 1-7 1-11 1-12 1-14
Novalgin amp 10x2cc 8-0;
Neo-salvarsan
15 30 45 60 75
0-14 0-15 1-1 1-4 1-8
N.A.B. 15 30 45 60
0-10 0-11 0-14 1-0
Mepharside 0·4 1-2; 0·6 1-4
Crooks V.D. 15 cc 3-0; 30cc 5-10
Ciba Entero Vioform Tab 20 2-12
" coramin liq 15 cc 3-10;
Glaxo Berin Bulb 10 cc 25 mg
[1-15; 50 mg 3-2; 100 mg 4-6
" Calci Ostelin 15 cc 3-4;
Vicks Veporub 1-6; Inhaler 1-4
Redoxan tab 20 1-8; 100 6-2;
" amp 2 cc 6 4-14; 50 36-0
" " 5 cc 3 4-4; Benarva
[Bulb 100 mg 3-14
Sandoz Cal Glu amp 10%
[10x5cc 8-8; 50 38-8
" Cal Glu amp 10%5x 10cc 5-2
[50 44-0
" " " " with Vit C 10%
[10x5 cc 10-12
" " " " 10%5x10 cc 6-4
Viganan tab 10 1-5; 20 2-10
Sil Vit Eng 3-4; Protergal oz 1-14
Adrenalin Chloride Eng oz 1-11
Chlorobutol " " 1-7
Aspirin Cryst Eng lb 4-6
Calci Hypo MB lb 4-10
Chlorobutol MB lb 11-8
Calci Lactate 2-8; Gluconate 3 2 lb
Chloral Hydras B.B. 5-14;
Calomel Italy lb 8-0
Ethyl Chloride Ger 100 grm 2-7
Pot Citras Eng 3-8
Soda Citras Eng lb 3-8
Ferri Ammon Cit Eng lb 4-8
Diuretin oz 2-1
Ext Ergot liq Ind 4 oz 7-0; lb 21-0
Soda Salycilas Ind 3-10; Eng
[3-12 lb
Sulphanilamide pow Eng lb 9-12
Creosote MB lb 7 0
Milk Sugar ICI 1-12 lb.
Mercurochrom 5 grm 0-10
" 10 grm 1-3; 25 grm 2-4
Acriflavin 5 grm 0-12; 10 grm 1-6
[25 grm 2-12
Hypo Syringe (S.N. Re. 1 More)
All Glass Jap
2 5 10 20 30
0-10 1-0 1-6 2-10 3-4
All G. German
1-4 2-4 2-10 3-8 —
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4-10 6-12 7-8 10-4
" Italy 3-8 4-14 7-0 9-4
" Boston USA 5-2 6-2 7-4 10-0
B.D. Liver Lock 7-12 11-8 13-8 17-4
Japan " " 2-0 3-0 4-0 6-0
Empty Metal case Japan
2-0 3-4 4-4 6-4
Hypo Record Needles
Down No 14, 16 Doz 5-4
Japan 1-14; German 2-0
M.D. 3-10; Down 4-6 Dz
All Glass Needles Luer Mount
Japan 2-8; German 3-6 Down 5-4 Dz
Hot Water Bag 3-4; Ice Bag 1-8
Bandages 1 2 3 4 5
0-13 1-10 2-6 3-2 3-14 dz.
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Tooth Forceps Universal 4-8
F.L. Durex Tin 1-12 Pkt 1-10 dz.
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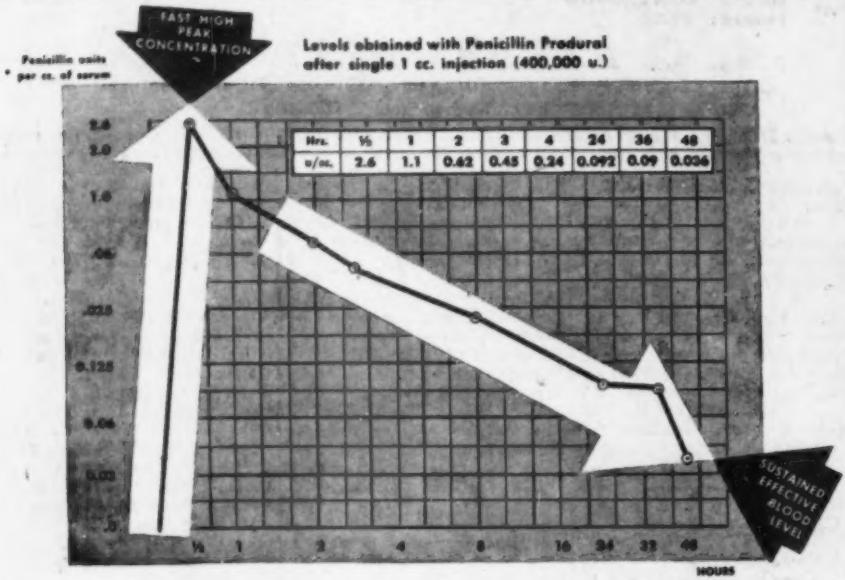
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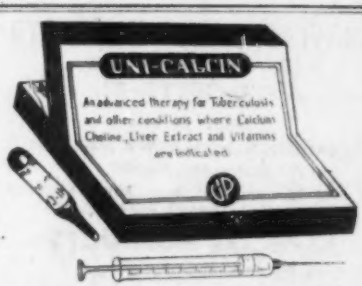
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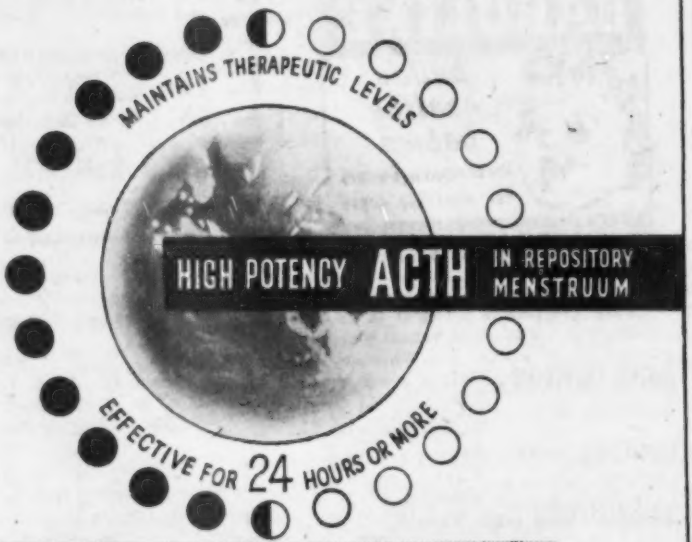


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Acriflavin 25grm. 2-8; 5grm. 0-12
Proflavin 500 grms. I.C.I. 26-0
Chloromycetin 12 Cap. ½ gr x 20 20-0
Auromycitin 8 Cap. 18-12
Terramycin 8 Cap. 17-4; 16 32-8
Combiotic (Strp & Pen.) 4-8
P. D. Camoquin Tab. 0-12; Combex
Penicillin G Crys. [10cc. 6-12
1 2 5 10 lacs
0-10 0-12-0 1-4-6 2-12 Merck
— 1-0-0 1-8-0 2-12 Pfizer
„ 0-9 0-11-6 1-3-6 2-4 Glaxo
Strepto c P.A.S. Lepetit 5-4
Dihydro Strepto 1grm. Glaxo 2-3
A & H 2-2; Pfizer 2-8; Merck 2-4
Procain Penicillin (Destaquin)
B.W. or A.&H Glaxo Pf.
3 lacs 1-0 — —
4 lacs 1-8 1-6-6 1-12
3 lacs x 10 cc. Oily Merck 9-0
Chloromycetin Liq. Bot. 16-4
Penicillin Skin Oint 1-2 Eye 0-11
„ Lorexgis 20 1-0 [2 lacs x 10 5-12
„ Tab. ½ lac 12 4-2 1 lac 7-12 PF.
P.A.S. Dumex Bayer Italy Rodia
„ 100 grm 10-4 12-8 4-6 6-0
„ 100 tab. Herts 5-14
„ „ Bayer 6-4; Italy 3-14
„ 250 Italy 9-8; 500 16-0
P.A.C. tab. 75 6-2; 250 17-4
Quinine Jap. 42-0; Stand 40-0
„ Holland. 49-0; Howds 51-0
„ oz Ind. 3-4; Howds 3-10
Q. Bihydro Amps. 100x10grsx2cc.
„ Ind. B.D.H. Evans B.W. P.D.
„ 17-8 22-8 22-8 35-0 40-0
„ 12-0 16-0 16-0 100x5gr.x1cc.
Euquinine Holand or Java 4-2
„ Roche 4-8;
Q. Tab. 2grx100 2-2; 5gr 4-0 How
„ „ 5 gr. 1400 How. 45-0
„ Bihydro 2grx100 3-0; 5gr 5-0 How
Pamaquinine 300 Tab. 0-12
Oral Tablets 1000 500 100
Asparine tab. 5-10 1-2
Mepacrine Eag. 10-4; ICI 12-12
Quinacrine MB 13-4; 7-0
„ USA. 5000 51-4; tin 1000 12-8
Ephedrin ½ Gr. 1000 6-12; Ind. 5-4
Yeast Tab. Eng. 6-12; Squibs 12-0
Soda „ nt „ 2-6; 100 0-12
Paludrin 1000 x 1 gr. x 25-0
„ 3 grm x 500 25-14; 496 27-8
Aminophylin Tab. 25 2-0 100 5-0
„ Amps. 6x2cc 5-0; 6x10cc. 5-10
Ext. Ergot. 4 oz 9-12 lb 35-8 Eng.
Phenobarbiton Tab. 1000x1gr. 14-0
Saridon tab. 10 1-7; 250 25-4
Potas Chloras IIb. 3-0; tab. 500 3-8
B.W. Morphia Sulph c Atro. 20 x
Indian M.S. 2-8 [½ gr. 5-0
P.D. „ „ „ 3-8
Morphia S. c Atro ½ gr x 12 amps. 5-8
Sulphamesothin amp. 25 x 0.3
[10/- box [tin
Multivitamin tab. USA 1000 20/-
Niacin tab. 25mg.x100 24/- doz.
„ 100 mg. x 100 37/- doz.
„ 50 mg. x 100 42/- doz.
Liver Ext. 4 U.S.P. 10cc Belgi. 3-0
Leucarson 4 gr. x 500 16/-
Riscohin tab. 10 1-12; 100 14-11
Redexon6x2cc. c 4-14; 50x2cc. 37-0
„ 3x5cc. 4-4; 25 x 5cc. 34-0
Merck H.T. Enstin ½ gr. or 1 gr. 3-12
Sulpha Tab. 1000 500
„ nilamide Eng. 10-4 5-4
„ guinidine „ 24-0 0-13
„ thiazole Eng. 36-0 18-8
„ mezathine (100 6-12) 30-0
„ diasine USA 77-0; MB 49-0
„ „Boots 42-0 500 BDH 500 44-0
Sulphatrone (100 9-12) 40-6
Sulphatriad MB (100 9-0) 43-8
Sulphonamide pwd. 1 lb. 10-8
„ cream 4 oz. Lilly 5-0 doz.
Gentian Violet Jelly 4 oz. Lily 4-8 „
Emetine amps. BDH ½ gr. x 12 7-8
„ 1 gr.x12 13-0; ½ gr. x 25 14-8
„ „ „ Endo 6 x ½ gr. 2-4
„ P.D. ½gr.x 6cc 6-10 1 gr. 10-12
B.W. ½ gr. 9-12; 1 gr. 8-0
Cibazol 250's 14-12; 20's 1-12
MB 760 28-0; MB 693 500's 41-0
Vit. B. Complex 10 cc 5-0; Belgium
First Aid Box 11-8 [10cc Bulb 3-12
Vitamin B tab. U.S.A. 500 1-12
Liver Ext. 10cc. 2 USP P.D. 3-12
„ „ 5 USP P.D. 8-0
„ Eng. 5 USPx10cc. 4-6 20cc. 8-4
Campolon 5x2cc. 5-8 25x2cc. 25-8
Cal. Glu. 10% x 10 cc. 100 14-0
Glucose Sol. 25%x25cc.x100 22-0
„ „ Thilo Germ. 50 amp. 20-0
Milk with Iodine 100 x 5 cc. 16-0
Cyclotropine I.V. I.M. Ger. 5-8 & Eag.
Atophanyl I.V. or I.M. 6-4 [4-12
Beris 25mg. x 10cc. 2-0 50mg. 3-2
Calci Ostelin 15cc. 3-4 [100mg. 4-6
NAB. 15's 0-10; 30 0-11; 45 0-13;
Nicotinic Acid 500 3-4 [·6 0-15
Neosalversan 0·15 ·30 ·45 ·60grs
(·75gm. 1-12) 1-1 1-2 1-4 1-8
Acetolarson Adult 6-4 Child 4-12;
Atebrine Bayer 15 0-11; 300 3-0;
PD Adrenalin in oil 3-6 [1000 14-0
Pituitrin 6x1cc. 11-0 ½ cc. 8-2P D
Distil Water 100 x 5 cc. 5-12
„ „ 10 cc. 8-0; 2 cc. 5-0
Sil.Vit. Eng. 3-8; Protargol 2-0
Ethyl Chl. 100grm. German 2-8
Sedasalg 4-0 lb. Santonine dr. 6-4
Thermometer Germ. 1-1; Jap. 0-12;
„ Zeal 2-8; USA 1-9; Eng. 1-9;
„ Hicks 4-0; Vibronawine 8-8
B.D. Stethoscope 22-8; Ger. 10-8
Plastic tubing 1-8; Rubber 0-12 yd
Erkameter 60-0; Aspirin 4-10 lb.
Detecto Weighing Machine 38-0
Saline Apparat. comp200c.c. 8-0
Wincarnis large 11-0 Hazola 10-12
Wall Thermometer Japan 2-8
Sandoz. Cal. Gluco „ Vit C
5 x 10 c.c. 6-4; 10x5 c.c. 10-8
B.W. Cal. Glu. 10% x 10 cc. 100 45-0
Sandoz „ „ „ „ 5 amps. 5-0
„ „ „ „ 5 c.c. x 10 8-8
„ 50 amp. 45-0 10c.c.x 20 19-0
Entrevisforn 20's 2-14; 100's 11-8
Abs. Cotton 1-0; Lint 3-6
Abs. Gauze 18 yds x 25" 4-12
„ „ 6 yds. 4-8 doz. [per inch
Bandages 4 yds. x 1" to 6" 0-12
„ Bandages 6 yds.x3" JJ 4-0 doz.
Scissors 1-6; Steriliser 6" 12-0
Hot water Bag 3-4; Ice bag 1-12
Stethoscope pouche plastic 2-0
Hypo. Syringe (S.N. Rs. 1 more)
A.G. Jap. 2 5 10 20 30cc.
0-10; 0-14; 1-3; 2-4 3-0
Italy 1-10 2-2 2-12 4-0 7-0
Germ. 1-2 1-8 2-8 3-0 7-0
Record Ger. 3-4 5-8 6-8 10-0 13-8
„ Comp. 6-4 9-0 11-4 — —
„ Italy 3-8 4-12 7-0 8-8 12-0
Boston 5-0 6-8 7-8 11-8 15-0
B.D. Luer 7-8 10-8 12-8 16-0 20-0
Japan „ 1-14 2-14 4-0 6-0 9-8
Italy B.case1-2 1-8 .2-0 2-8 —
Metal case Ind. 50cc. 5-12
Bakelite case 1-8 2-12 3-0 —
Hypo Syringe 50 cc. S.N.
Jap. 5-8; Italy 10-8; Germ. 9-12
B.D. Luer Lock 28-8; Jap. 16-0
Record Ger. 23-0; Italy 17-8
Record Needle (Perfectum 5-4)
Jap. Germ. Star. D.B. 14, 16
2-0 2-0 3-12 4-4 5-4 Dz.
All Glass Needles Luer Mount
Jap. Ger. D.B. B.D.
2-8 3-4 5-8 9-8 doz.
Atebrin Amp 3 grm x 2 2-8 25 17-0
„ 0.1 grm 6 2-6 [Tooth Forcep 4-8
Camphor-in-Oil 3 gr. x 1c.c x 100
Amyl Nit. Cap. 2-0 [Cipla 3-8
Omnopon Amps with Needle
Nivaquin 10 1-12 [Tube 1-0
F.L. Durex Tin. 3-0 doz. Pkt. 2-0
Ear, Metal Syringe 2cc. 5-8;
Waterbury Co. 5-0 bot. [4 oz. 6-12
Vit. B12 Glaxo 20 mic. 6x1cc. 3-4
„ 50 mic. x 6 1cc. 5-8; 5cc. 3-12
Vitamin K. amp. 100 B.W. 18-0
Normal Saline 100 x 5cc. 7-0
Oil Chiscpodium oc. 6-0 [10cc. 9-12
Rubber Gloves 7½" or 8" 1-0
Disp. Scale Nick. 5-0; Brass 4-0
Argyrol Original 7-0


**Morphia Tartarate Amp. Squibbs 5 x 1·5 cc. x ½ gr. 0-10 box.**

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Vol. XLIX, No. 9 Regd. No. 9, M. 429 SEPTEMBER, 1952

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MEASLES
MUMPS
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URINARY INFECTIONS
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